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CLINICAL AND SURGICAL ASPECTS OF SPREADING PERITONITIS COMPLICATING ACUTE PERFORATIVE APPENDICITIS*

JOHN O. BOWER, M.D.

Director of Research of The Foundation for Clinical and Surgical Research
Philadelphia, Pennsylvania

IN Philadelphia in 1922 a preliminary investigation revealed that the mortality of acute appendicitis was very high.†

A survey of 1,002 clinical records of patients operated upon for acute appendicitis in two of the largest hospitals disclosed the fact that the causes for the high mortality, delay in hospitalization and the administration of laxatives, are controllable. Through the efforts of Andrew A. Cairns, M.D., then Director of the Department of Public Health of Philadelphia, a city-wide survey of the clinical records of acute appendicitis cases in twenty-eight hospitals was made in 1930. The results obtained confirmed the findings of the initial survey and supplied statistics and facts with which a prophylactic campaign was inaugurated. The results of this continued education of the public show a consistent reduction of the mortality as shown by five subsequent surveys.

During the ten years the prophylactic campaign has been in progress surgical research on spreading peritonitis, the cause of over 92 per cent of the deaths, has been conducted.

In 1933 the Medical Society of the State of Pennsylvania appointed a Commission for the reduction of the mortality of acute appendicitis and a prophylactic campaign has been since conducted throughout the state by its members, one from each of the twelve Councilor Districts with their District and County committees, numbering over 300 physicians and surgeons.

TABLE I. MORTALITY OF ACUTE APPENDICITIS IN PHILADELPHIA

Six Surveys

Year	No. of Cases	No. of Deaths	Mortality Per Cent
1928-29	5,121	306	5.97
1930	3,095	149	4.01
1931	3,142	138	4.39
1932	3,546	122	3.44
1933	3,783	134	3.54
1937	4,186	102	2.44

The first Pennsylvania state-wide survey was completed April 1, 1940.

The results of the Philadelphia prophylactic campaign, surgical research, and now the results of the Pennsylvania campaign, indicate that:

1. Patients do not die from acute appendicitis; they die from spreading peritonitis.
2. Patients with appendicitis-peritonitis should be managed by surgeons who have some knowledge of the immunologic aspect of the problem.
3. The prophylactic campaign is the surest method of reducing the mortality.
4. In considering a plan for prevention of deaths from appendicitis-peritonitis the prophylactic removal of the appendix in the very young must not be overlooked, as there is no mortality other than the unavoidable catastrophe that can be attributed to the operation *per se;* approximately one in 1,000.

In Table II are listed the nine principal causes of death in the United States, with the number that die annually from each, and the age group in which most deaths occur.

*Presented before the annual meeting of the Minnesota State Medical Association, Rochester, Minnesota, April 24, 1940.

†Dr. Hoffman of the Prudential Life Insurance Company had published a national statistical study which showed that the mortality of acute appendicitis in the United States was (1) 30 per cent higher than Scotland, (2) 51 per cent higher than Germany, (3) 73 per cent higher than England and Wales, (4) 76 per cent higher than New Zealand, (5) 83 per cent higher than Italy, and (6) 115 per cent higher than Irish Free State.

TABLE II. PRINCIPAL CAUSES OF DEATH IN UNITED STATES

Disease	Number of Deaths Annually	Group in Which Most Deaths Occur
Heart	371,675	70 – 80
Cancer	149,126	60 – 70
Nephritis	126,879	70 – 80
Pneumonia—		
Lobar	67,778	50 – 60
Bronchial	51,600	75 – 85
Accidents	110,249	20 - 30
Apoplexy	103,560	70 – 80
Tuberculosis	71,527	20 – 30
Diabetes	30,406	65 – 75
Appendicitis	20,000	10 – 20

Acute appendicitis is a disease of youth. The average age is twenty-three years; death occurs at the average age of thirty-four. It is estimated that over 18,000 die each year in the United States, 3,240 before the age of twenty.

Deaths from appendicitis-peritonitis can be eradicated by definite instruction in the schools. As shown by the following table, over 40 per cent of cases occur between the ages of ten and twenty and over 53 per cent between one and twenty.

In Philadelphia the high school students have been instructed for ten years, in Pennsylvania for only five. Notice the difference in the mortality (Table IV).

TABLE III. MORTALITY IN DECADES

40.47 Per Cent of Cases in 2nd Decade

	MALE			FEMALE			
Ages in Decades	Recoveries	Deaths	Mortality Per Cent	Recoveries	Deaths	Mortality Per Cent	Total
1-10	1,236	53	4.11	1,045	40	3.68	2,374
11-20	3,624	76	2.05	4,227	55	1.28	7,982
21-30	2,647	64	2.36	2,280	24	1.04	5,015
31-40	1,121	55	4.67	730	17	2.27	1,923
41-50	641	69	9.71	405	36	8.16	1,151
51-60	281	51	15.36	235	31	11.65	598
61-70	112	31	21.67	101	31	23.48	275
71-80	31	14	31.11	16	5	23.81	66
81-90	5	6	54.54	2	0	0	13
91-100	1	0	0	0	0	0	1
Totals	9,699	419	4.14	9,041	239	2.57	19,398

All Cases, Average Age—23.4 years; Recoveries—23 years; Deaths—34.2 years.

TABLE IV. DELAY—MORTALITY 1937

	PHILADELPHIA			PENNSYLVANIA		
	No. Cases Recovered	No. Cases Died	Mortality Per Cent	No. Cases Recovered	No. Cases Died	Mortality Per Cent
Admitted Within 24 hours	2,059	16	.77	9,061	124	1.36
Admitted Between 25-48 hours	1,179	35	2.97	5,792	223	3.85
Admitted Between 49-72 hours	342	14	4.09	1,778	105	5.90
Admitted After 72 hours	606	37	6.10	2,767	206	7.44
Totals	4,186	102	2.44	19,398	658	3.39

TABLE V. DELAY AND MORTALITY—APPENDICITIS-PERITONITIS

	Year	Admitted within 24 Hours Per Cent	Perforations Per Cent	Spreading Peritonitis Cases Admitted Per Cent	Total Cases Mortality Per Cent
Philadelphia	1928-29	32.08	42.96	13.54	5.97
	1930	30.31	35.84	15.25	4.01
	1931	29.60	35.30	15.62	4.39
	1932	32.40	29.02	12.80	3.44
	1933	30.06	26.25	12.03	3.54
	1937	49.19	12.21	8.26	2.44
Pennsylvania	1937	46.71	17.64	12.07	3.39

Table V shows that by earlier operation and by increasing the percentage of those admitted to hospitals in the first twenty-four hours the percentage of perforations and the mortality can be reduced.

Table VII shows the results of laxatives on the mortality of spreading peritonitis.

TABLE VI. ACUTE APPENDICITIS—APPENDICITIS-PERITONITIS

Results of Laxative Administration

	No. of Patients	No. of Deaths
No Laxative Administered	1,733	27 (1 in 64)
One Laxative Administered	2,723	135 (1 in 20)
More than One Laxative Administered	839	71 (1 in 12)

TABLE VII. SPREADING PERITONITIS MORTALITY

Results of Laxative Administration

	No. of Patients	No. of Deaths	Mortality Per Cent
No Laxative Administered	141	23	16.31
One Laxative Administered	567	128	22.57
More than One Laxative Administered	260	69	26.54

TABLE VIII. MORTALITY OF ACUTE APPENDICITIS IN PHILADELPHIA

Year	No. of Cases	No. of Deaths	Mortality Per Cent	No. of Lives Saved
1928-29	5,121	306	5.97	
1930	3,095	149	4.01	36
1931	3,142	138	4.39	50
1932	3,546	122	3.44	89
1933	3,783	134	3.54	92
1937	4,186	102	2.44	148
	22,873	951	4.15	415

High school students are warned about the dangers of delay in hospitalization and the administration of laxatives. In all, 110,000 high school students in Philadelphia, and 630,293 in Pennsylvania were told that the mortality of appendicitis-peritonitis increases with each twenty-four hours.

We warned them about laxatives—when they develop acute abdominal pain Nature stops the movements of the intestines so that the inflammation will remain localized. If they take laxatives the intestines are thrown into activity which may result in rupture and appendicitis-peritonitis.

APPENDICITIS WARNING

For pain in your stomach—
Don't take a laxative or physic.
Don't take anything by mouth.
See your doctor immediately.
If he says it's appendicitis and advises operation—
Don't delay going to the hospital.
Laxatives and delay frequently cause spreading peritonitis and death.
This warning is published by The Medical Society of the State of Pennsylvania.

Each high school student should be given a warning sticker with the request that he take it home and affix it to the family medicine cabinet where it can be read by the other members of the family. They should be told that when anyone develops abdominal pain some organ inside the abdomen is not functioning properly. Pain is a warning. The first symptom of acute appendicitis is general abdominal pain which later centers in the lower right side, the region of the appendix.

High school students are being taught to recognize acute appendicitis. Does this plan pay in dividends of lives saved?

An attack may not be accompanied by nausea

or vomiting. There may not even be a rise in temperature. The most important thing to have done is a blood count. When the appendix becomes inflamed, tenderness develops and Nature stops movements of the intestine because of the urgency of keeping the infection confined to the appendix.

In the proposed plan to educate the high school student to recognize acute appendicitis, where does the family physician fit into the set-up? He has four vital functions: (1) he must establish or disprove his patient's "homemade diagnosis;" (2) if he concurs in the diagnosis he must see to it that his patient is placed in the hands of a surgeon—not an operator; (3) if he does not agree with the "homemade diagnosis" he must determine definitely what is wrong with his patient; and (4) he should participate in the prophylactic campaign.

Even when a youngster is intelligent enough to describe the character of the initial abdominal pain and can demonstrate with a finger the maximum point of tenderness, and even though in 90 per cent of instances he tells the physician what is wrong with him—a complete examination, which includes a rectal examination, should be made. Catastrophes have occurred because physicians have not examined patients' abdomens, and catastrophes have been prevented by youngsters refusing to take laxatives ordered by family physicians. The family physician must consider himself a consultant if fewer mistakes in diagnosis are to be made.

The selection of a surgeon by the family physician is important. It must be remembered and reiterated that an appendectomy may be the simplest or the most difficult of operations. I have mentioned that the family physician should consult a surgeon and not an operator. The difference is this: the operator operates; the surgeon manages. The operator can and does successfully remove intact appendices but he treats the perforated and the non-perforative appendices alike. The perforated appendix requires management. It requires the best the hospital affords. Why? Because over 92 per cent of the mortality is in this group. One of every four patients admitted to hospitals with perforated appendices dies.

When patients die following operation, the operator thinks they die of *what they had when he operated upon them,* and not *what he did at operation.* The operator uses one kind of incision, one kind of anesthetic, and one kind of operation. He prides himself on how fast he can remove an appendix. He reports his successes in the form of appendiceal mortality, covering up the true mortality of spreading peritonitis by a camouflage of non-ruptured appendices, which have no mortality.

The family physician is the logical person to tell the public—which begins with his patients—about the prophylactic campaign to prevent death from appendicitis-peritonitis. He should use the warning stickers and he should be willing to speak before high school assemblies and lay organizations.

Finally, the family physician and surgeon should attempt to convince their State Health authorities that appendicitis-peritonitis is a public health problem; that it is as much an immunologic problem as the seven most frequent communicable diseases, and that the same amount of money should be spent in a publicity campaign as is spent in immunizing children against measles, diphtheria, typhoid, et cetera.

TABLE IX. PUBLIC HEALTH ASPECT OF APPENDICITIS-PERITONITIS

	Minnesota	Pennsylvania
Population—1937	2,652,000	10,158,000
No. High School Students, Junior and Senior	175,007	631,293
Deaths from Seven Most Frequent Communicable Diseases	223	945
Deaths from Appendicitis-Peritonitis	307	1,085

There is no mortality from acute appendicitis. Spreading peritonitis causes 92.4 per cent of the so-called appendicitis deaths. Patients do not die of acute appendicitis; they die of spreading peritonitis.

The facts in Table X were obtained from the abstracts of 38,085 clinical records.

TABLE X. MORTALITY IN APPENDICITIS AND APPENDICITIS-PERITONITIS

	Number of Cases	Deaths
Acute Appendicitis	28,235	104 (1 in 271)
Appendiceal Abscess	4,935	101 (1 in 49)
Spreading Peritonitis	4,915	1,302 (1 in 4)
Total	38,085	1,507 (1 in 25)

TABLE XI. MORTALITY IN ACUTE APPENDICITIS AND SPREADING PERITONITIS

Philadelphia Year	Acute Appendicitis			Spreading Peritonitis		
	Number of Cases	Number of Deaths	Mortality Per Cent	Number of Cases	Number of Deaths	Mortality Per Cent
1928-29	5,121	306	5.97	698	237	33.95
1930	3,095	149	4.81	472	124	26.25
1931	3,142	138	4.39	491	120	24.44
1932	3,546	122	3.44	457	101	22.10
1933	3,783	134	3.54	455	112	24.61
1937	4,186	102	2.44	346	89	25.72
Total	22,873	951	4.15	2,919	783	26.82
Pennsylvania 1937	19,398	658	3.39	2,342	608	25.95

That patients die from spreading peritonitis, not appendicitis, is further corroborated by the fact that 22,000 clinical records previously abstracted in six Philadelphia surveys show the same results within a fraction of a per cent.

Patients admitted to hospitals with a diagnosis of acute appendicitis with an intact appendix do not die from this disease, statements on their clinical records to the contrary. These patients die a catastrophic death. Of the 658 deaths, 118 were catastrophes — twenty-nine unavoidable, eighty-nine avoidable.

The twenty-nine unavoidable catastrophes represent the hazards coincident to any group of comparable size. Surgeons accept them as inevitable. The majority occurred in the later decades of life. Only seventeen of the twenty-nine were unruptured. This number (17) represents the total mortality of the 16,046; only one in 944 died.

In the avoidable group, a total of eighty-nine deaths, errors in surgical management were responsible for seventy-one, errors in diagnosis for eighteen. Patients' relatives, as well as the surgeon, know when a death following operation is unavoidable. Explanations are unnecessary. Catastrophic deaths in the avoidable group, however, are difficult to explain, not only to the family, but to the family physician as well.

Errors in Diagnosis.—In this presentation it will be impossible to consider each of these in detail. Pneumonia, the most common error, occurs most frequently in the juvenile. A correct diagnosis can be made at times only by exhausting every possible means at our disposal; careful history, leukocyte count, x-ray, consultation.

TABLE XII. CATASTROPHIC DEATHS UNAVOIDABLE

Embolism	11
Cardiac Dilatation (so-called)	6
Coronary Disease	5
Myocarditis (so-called)	2
Thrombosis	1
Atelectasis	1
Apoplexy	1
Pulmonary Infarct	1
Uremia	1
Total	29

TABLE XIII. CATASTROPHIC DEATHS AVOIDABLE

Errors in Diagnosis		Errors in Management	
Pneumonia	8	Appendix Ruptured on Removal	58
Intestinal Obstruction	2	Anesthetic Deaths	8
Ruptured Cecum	1	Hemorrhage	4
Ruptured Duodenal Ulcer	1	Postoperative Venoclysis	1
Ruptured Gastric Ulcer	1		
Acute Salpingitis	1		
Typhoid Fever	1		
Diabetes	1		
Myelogenous Leukemia	1		
Otitis Media	1		
Total	18		71

Usually operation is decided upon because of rigidity. If rigidity is the deciding factor favoring operation then operation should be delayed, because an extensive rigidity is usually due to spreading peritonitis resulting from perforation. Delay for deliberation or deliberate delay until a definite diagnosis can be established is justifiable. The importance of the rectal examination should be remembered. It is far more important in a suspected acute surgical abdomen in children than a simple abdominal examination. Immediate operation in the presence of spreading peritonitis has a mortality rate of 27 per cent. The death rate of patients operated upon for suspected acute appendicitis, but who actually have pneumonia, approximates 75 per cent.

Intestinal obstruction is unusual but it occurs about once in 500 cases; an appendiceal abscess, partially blocking coils of intestine, is responsible. Deaths are due to spreading peritonitis, induced by the operator when he blindly inserts his hand into the peritoneal cavity and ruptures an abscess, in attempting to deliver the obstructed loop. It is better surgery to enlarge the incision.

Errors in Management. — Hemorrhage — a death has occurred from post-operative hemorrhage following removal of an acutely inflamed appendix in about 1 in 4,000 cases. This can be prevented by first ligating the stump and then inverting it with a very fine chromic catgut suture. If the suture is crossed at the mesenteric junction the purse string will compress a branch of the posterior ileocecal artery and the proximal branch of the appendicular artery, which are usually responsible for the bleeding.

Anesthetic Deaths.—Satisfactory notation regarding the anesthetics administered were obtained in only 14,894 of the 19,398 records abstracted. There were eight anesthetic deaths. The average age of those who died was forty; on admission to the hospital the average temperature was 99.4, pulse 105. Five of the eight had unruptured appendices; three had peritonitis. Six of the eight died on the operating table.

The results of this survey show that 3,640 patients were given ether without a death; 7,307 were given gas alone or in combination, with five deaths; 581 were given avertin alone or in combination, with one death.

A spinal anesthetic was given to 2,111 patients. Two died. One was given spinocaine. The other anesthetic was not mentioned. Local anesthesia was used in only 114 instances. There were no deaths. Ether is still the safest anesthetic.

TABLE XIV. ACUTE APPENDICITIS—APPENDICITIS-PERITONITIS ANESTHESIAS

14,894 Administered—8 Deaths
6 General, 1 Spinal, 1 Unknown

Anesthetic	Number	Deaths
Nitrous Oxide Oxygen-Ether	6,582	4
Nitrous Oxide Oxygen	725	1
Avertin, alone and combined	581	1
Veneth. Epival, etc.	45	0
Ether	3,640	0
Ethyl Chloride and Ether	640	0
Cyclopropane	237	0
Ethylene	124	0
Ethyl Chloride	95	0
Total	12,669	6

Spinal Anesthetic	Number	Deaths
Not Mentioned	762	1
Pro-Novocaine	538	0
Pantocaine	273	0
Neocaine	269	0
Spinocaine	230	1
Metycaine	32	0
Anesthetol	5	0
Nupercaine	2	0
Total	2,111	2
Local Anesthetic		
Novocaine	114	0

TABLE XV. APPENDICITIS-PERITONITIS—CLINICALLY APPLIED PATHOLOGY

LOCALIZED MASS—	Appendix, serous coat intact, partially or completely covered with lymph or omentum—or both. (Fig. 1)
LOCALIZING PROCESS—	Appendix, serous coat perforated. Perforation sealed with lymph, omentum or intestine. (Fig. 2)
	Beginning abscess surrounded by omentum or intestine—or both.
LOCALIZED PROCESS—	Frank abscess. (Fig. 3)
SPREADING PROCESS—	Spreading peritonitis. (Fig. 4)

Rupture of acutely inflamed appendices at operation was responsible for the greatest number of avoidable catastrophic deaths. The spreading peritonitis which followed was so virulent that it will be considered under the caption "Hyper-

TABLE XVI. ACUTE APPENDICITIS—APPENDICITIS-PERITONITIS
DRAINAGE—MORTALITY

	Acute Appendicitis		Appendicitis-Peritonitis	
	Not Drained	Drained	Not Drained	Drained
No. of Cases	14,136	1,840	126	3,301
No. of Deaths	0	6	74	544
Mortality Per Cent	0	.33	58.73	16.48

acute Perforative Group."—This catastrophe occurred once in each 200 patients operated upon with intact appendices.

Two years ago a clinical pathological classification of Acute Appendicitis-Peritonitis was published, based on what is found by the surgeon at operation—not what he leaves in the peritoneal cavity after operation or what the pathologist finds at postmortem. The pathological process can be described as either an inflammatory mass or an inflammatory process.*

Let us consider first the localized mass. The mortality is 1 in 169. Of the 15,976 clean cases, 14,136 were not drained. There were no deaths. 1,840 were drained and 6 died.

These surgeons should question their surgical judgment in draining non-perforative cases when they study the results shown in the drainage table above. If a surgeon at operation finds a perforated appendix, if he is doubtful as to whether or not the serous coat is intact, or if hemorrhage cannot be controlled by ligature, the insertion of drains is justifiable. It is not only definitely unnecessary but undesirable to insert drains of any kind into the peritoneal cavity to remove serous, serosanguinous or purulent fluid if the serous coat of the appendix is intact, because this fluid has a definite protective value. It is one of the earliest manifestations of Nature's attempt to develop resistance to microörganismal invasion. Titration of this fluid against the toxin of the *Clostridium welchii* shows that it contains antitoxin in 33.33 per cent of cases. The antitoxic titre of this fluid is highest in patients suffering from a non-perforative recurrent attack, in which the serous coat of the appendix had ruptured in a previous attack, but was subsequently sealed.

The amount of antigen absorbed during the average attack of acute inflammation of the appendix is so small that antitoxin appears in the blood serum in only 22 per cent of instances; but following perforation, antigen in larger amounts is absorbed, and if the patient lives, antitoxin is found in the blood serum of over 70 per cent of instances. Another important reason for withholding drains in the non-perforative cases is that complications, such as peritoneal infections, resulting in fecal fistulas and intestinal obstruction occur more frequently and the morbidity and mortality are increased over the non-drained cases.

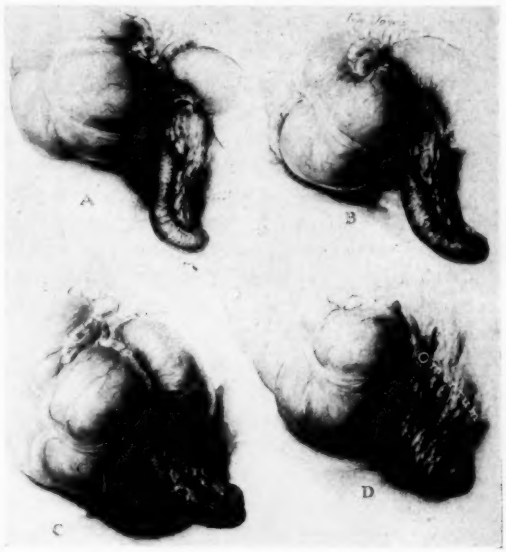


Fig. 1. *Localized Mass.* The inflammatory mass may be an acute catarrhal, ulcerative, suppurative, or gangrenous. The removal of any of these will result in cure. Because of catastrophic deaths the chances of dying are one in 169.

In seventy-one hospitals of the 181 surveyed, 118 operators did not drain the peritoneal cavity following removal of a perforated appendix in 126 patients and seventy-four, or 58.73 per cent, died.

Those who have not instituted drainage of the peritoneal cavity in the presence of a perforated

*See Clinical-Pathological Classification. Am. Jour. Surg., 45: 66, (July) 1939.

TABLE XVII. APPENDICITIS-PERITONITIS CLOSED WITHOUT DRAINAGE

	No. of Surgeons	No. of Patients	No. of Deaths	Mortality Per Cent
Acute Intact, Ruptured on Removal	32	37	36	97.27
Spreading Peritonitis	34	35	24	68.57
Localizing Process	35	36	14	38.89
Abscess	17	18	0	0.00
	118	126	74	58.73

appendix or following its removal have, undoubtedly, read or heard of some operator advocating this procedure. They justify themselves by reasoning that appendices rupture and abscesses develop which are absorbed. It is true that this is not uncommon, but such patients recover not so much because pus is absorbed, but because toxins are absorbed slowly. Nature accomplishes this by the formation of a pyogenic membrane within the wall of which microörganisms become attenuated. Monomicrobic infections of the peritoneum are the most virulent and do not tend to localize or form this protective membrane. Fortunately, in the majority of instances, more than one organism is responsible for the peritonitis secondary to acute perforative appendicitis. In over 90 per cent of instances, the infection is not only polymicrobic but both anaërobes and aërobes are present. It is possible that this is responsible for the low virulences of the microorganisms present in the appendiceal abscess, simple drainage of which rarely results in death.

Table XVII shows the number of patients and deaths with the percentage mortality of those operated upon for intact appendices which ruptured on removal but were not drained; patients operated upon for spreading peritonitis; the localizing process, and abscess.

There is no operation for lesions involving organs within the peritoneal cavity of man, in which streamlined-brain-management pays as high dividends in deaths as operation for the distended appendix which ruptures on removal where the operator closes the wound without drainage. The mortality is high because of the rapid absorption of virulent antigen.

The percentage mortality of each of the lesions in Table XVII diminishes with the development of local and general immunity.

Approximately 11 per cent of the 1,705 surgeons failed to place or failed to leave out drains in the peritoneal cavity when the diametrically opposed procedure was indicated. This observation is in part responsible for the term "appendicitis-peritonitis." It is used to describe the peritonitis following rupture of the appendix and does not refer to the peritoneal reaction preceding perforation, which results in the extravasation of serous, mucoserous or serosanguinous fluids. The other reason for this term is that surgeons have camouflaged their spreading peritonitis mortality under the so-called acute appendicitis mortality. The mortality of the twelve councilor districts of Pennsylvania shows how this may occur.

The mortality of acute appendicitis and spreading peritonitis in Councilor District No. 1 is low. Councilor District No. 12 ranks second in appendiceal but eighth in spreading peritonitis mortality. Table XVIII is interesting in showing the results of the prophylactic campaign. The mortality is highest in those districts where the high schools have not received the educational talks.

The rupture of a membrane several microns in thickness is responsible for the high mortality. The percentage mortality depends upon when and how this membrane is ruptured.

The hyperacute perforative group shows what happens when perforations occur early and are accompanied by peritoneal trauma. Fifty-eight patients, average age 25, normal health, abdominal pain, death in 216 hours—tragedy condensed in 14 words. That twelve of the seventy operated upon lived, that 80 per cent of those that died received laxatives, that everyone did his utmost to save those that died must be remembered. But if surgeons responsible for these patients' lives are to profit, then we must not forget that while these patients had every reason to live—average age twenty-four, average temperature 100, average pulse 104—they were operated on forty-eight hours after onset of symptoms and every one of the seventy appendices ruptured on removal.

TABLE XVIII. COMPARISON OF MORTALITY BY DISTRICTS

Councilor District	Population	Students	Appendicitis Mortality			Spreading Peritonitis Mortality		
			Cases	Deaths	Mortality%	Cases	Deaths	Mortality%
1	2,497,029	132,796	5,084	122	2.39	711	116	16.32
12	613,650	45,966	1,534	42	2.73	136	39	28.67
2	627,966	37,420	1,514	44	2.91	185	40	21.62
7	231,306	15,434	581	17	2.93	51	12	23.52
6	366,542	26,483	895	27	3.02	115	26	22.61
11	766,330	51,857	1,159	36	3.11	137	35	25.54
4	446,165	29,494	1,250	43	3.44	77	38	49.35
8	439,303	31,124	898	33	3.67	106	28	26.42
9	385,044	25,611	647	24	3.71	40	22	55.00
3	543,890	36,546	1,219	48	3.94	158	41	25.94
5	797,700	50,825	1,439	66	4.58	168	60	35.71
10	1,916,425	147,737	3,178	156	4.91	458	151	32.96
Totals	9,631,350	631,293	19,398	658	3.39*	2,342	608	25.96**

*3.39 is misleading because it includes the deaths due to peritonitis.
**25.96 is definite—it shows what has happened to 608 of the 2,342 patients who developed a spreading inflammatory process of the peritoneum.

TABLE XIX. HYPERACUTE PERFORATIONS—INDUCED SPREADING PERITONITIS

TOTAL			AVERAGE				
				On Admission			
No. of Patients	Deaths	Mortality Per Cent	Age	Temp.	Pulse	Onset Symptoms to Operation	Operation to Death
70	58	82.86	24	100	104	48 hrs.	168 hrs.

TABLE XX. THE SPREADING PROCESS

TOTAL			AVERAGE				
				On Admission			
No. of Patients	Deaths	Mortality Per Cent	Age	Temp.	Pulse	Onset Symptoms to Operation	Operation to Death
1,118	26	24.06	22	101.4	118	69 hrs.	144 hrs.

This group of patients is unique, not only because of the startling death rate, 82.86 per cent, but never before, to the writer's knowledge, has it been possible to analyze and report at one time so large a number of cases where the period of time between perforation and death has been accurately recorded. The abstracts tell exactly when, how, and why perforation occurred in these seventy cases. Rupture occurred on an average of forty-eight hours after onset of symptoms. Some of them ruptured as delivery was almost completed. Most of these patients recovered; only the peritoneum adjacent to the incision became infected. The majority, however, were ruptured in the peritoneal cavity in various locations. The appendix was frequently abnormally situated and difficult to locate because of poor visibility. Without exception, the peritoneum was literally prepared for the rapid absorption of toxins. Every move of the surgeon's hand was attended by trauma or death of endothelial cells with consequent exposure of lymph and arterial capillaries.

The average time between perforation and death was 168 hours. Some patients died so quickly that their abdominal walls were rigid at death. Distention, the usual accompaniment of the spreading peritonitis deaths, did not have time to develop. Most of them developed hyperpyrexia almost immediately after operation, and an uncountable pulse within forty-eight hours. Many of them never regained consciousness after operation and died in a toxic delirium.

These patients were operated on twenty-nine hours earlier and died seventy-two hours sooner

TABLE XXI. THE LOCALIZING PROCESS

TOTAL			AVERAGE				
				On Admission			
No. of Patients	Deaths	Mortality Per Cent	Age	Temp.	Pulse	Onset Symptoms to Operation	Operation to Death
1,154	281	24.35	30	99.7	100	98 hrs.	216 hrs.

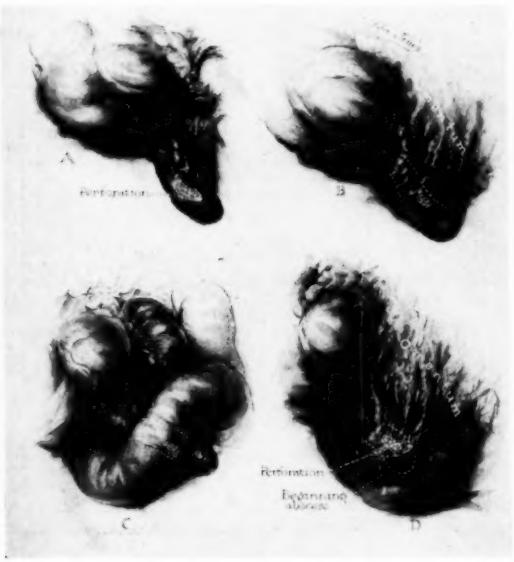


Fig. 2. *Localizing Process.* The localizing process, the spreading process, the localized mass are stages of a peri-appendiceal inflammatory process. All inflammatory processes of the peritoneum following rupture of an appendix are primarily spreading. As soon as the serous coat becomes devitalized permitting egress of microörganisms and their toxins, the contiguous peritoneum becomes involved. If the perforation is not large and laxatives have not been administered, the protective mechanism attempts to close the perforation by plastic exudate, omentum or intestine. This is the localizing process. When operated upon the localizing process is converted into a spreading process and one in four dies.

than those with the localizing process. Why? Because the vulcanizing plastic-lymph patch was blown off or the cemented intestine or omentum was detached by laxative induced peristalsis.

The acute perforation which results in fulminating peritonitis within four to twelve hours after onset of symptoms is not common. The subacute perforation, however, occurs frequently. More than twenty-four hours usually elapse between onset of symptoms and perforation. During this time—the preperforative state—the cells in the tissues in proximity to the potential perforative site undergo changes which are protective. This is local tissue immunity.

In all, 1,118 patients, average age twenty-two, were operated upon sixty-nine hours after onset of symptoms with a temperature of 101.4, pulse 118, and 269, or 24.6 per cent, died on an average of 213 hours after onset of symptoms.

Of the 658 deaths, 621 were the result of spreading peritonitis. The records show that fifty-eight of these 621 were induced at operation—hyperacute group. What about the others? Let us consider the localizing process.

Of the 19,398 cases, 1,154 were admitted with subacute perforations — localizing processes. These 1,154 patients, average age of thirty, were operated on ninety-eight hours after onset of symptoms with an average temperature of 99.7 and a pulse of 100, and 281, or 24.35%, died 314 hours after onset of symptoms.

Why should the mortality of this process be as high as that of the spreading process? Because of time of operation and what the surgeon did at operation. Time as it relates to the out-

TABLE XXII. CLINICAL PATHOLOGY CLASSIFICATION—MORTALITY

Pathology at Operation	Number of Cases	Per Cent of Cases	Number of Deaths	Per Cent Mortality
Localized Mass	16,046	82.72	95	.06
Spreading Process	1,118	5.76	269	24.06
Localizing Process	1,154	5.95	281	24.35 ?
Localized Process	1,080	5.57	13	1.20
	19,398	100.00	658	3.39

TABLE XXIII. THE LOCALIZED ABSCESS

TOTAL			AVERAGE				
				On Admission			
No. of Patients	No. of Deaths	Mortality Per Cent	Age	Temp.	Pulse	Onset Symptoms to Operation	Operation to Death
1,080	13	1.20	30	100.1	99	123 hrs.	235 hrs.

come of these patients will be considered under antigen—antitoxin and appendicitis. How, "what was done at operation" affects the mortality is shown in Table XXII. Has surgery failed in its purpose if individuals aged twenty-seven, with temperature of 100.2 and a pulse of 99, die after operation? Was something done which should not have been done at operation?

Does the explanation graphically shown in Table XXII seem reasonable?

The localizing process can develop into a spreading process or a localized abscess. A large percentage of localized abscesses were previously localizing processes. The mortality in the 1,080 with localized abscesses is 1.20 per cent, or one in eighty-three. *When appendices are removed in the presence of a localizing process the mortality is the same as the spreading process—one in four.*

How many of these 281 deaths were induced? The majority of these patients died with hyperpyrexia, tachycardia and distention, much the same as the hyperacute, except that delirium was less frequent and they lived forty-eight hours longer after operation.

Did time of operation and what was done at operation have anything to do with these patients' deaths? Is there any significance attached to the fact that both in the localizing and spreading group the mortality is one in four?

In the localized process group, 1,080 patients, average age thirty, were admitted to hospitals with an average temperature of 100.1 and pulse of 99, and thirteen, or 1.02 per cent, died. Those that died lived 235 hours (almost ten days) after operation.

From the standpoint of mortality this group approximates more closely than any other the acute intact group. There are comments and criticisms that can be made regarding the thirteen deaths, but we believe the surgeons who read this report will profit more by a comparative study of the results obtained in the management of 345

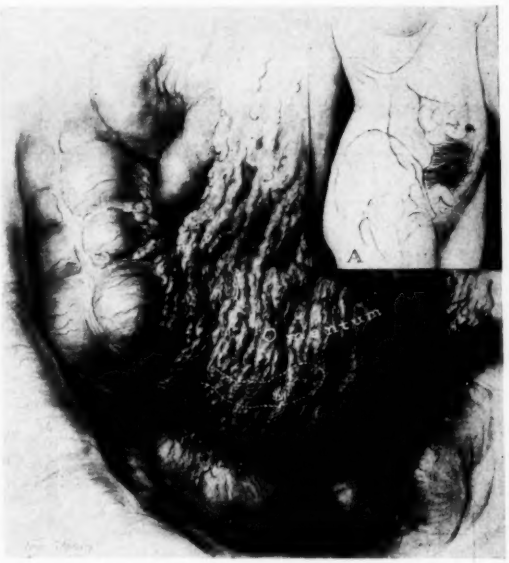


Fig. 3. *Localized Process.* The localized process—the appendiceal abscess—the end-result of the localizing or the spreading process; one in eighty-three die.

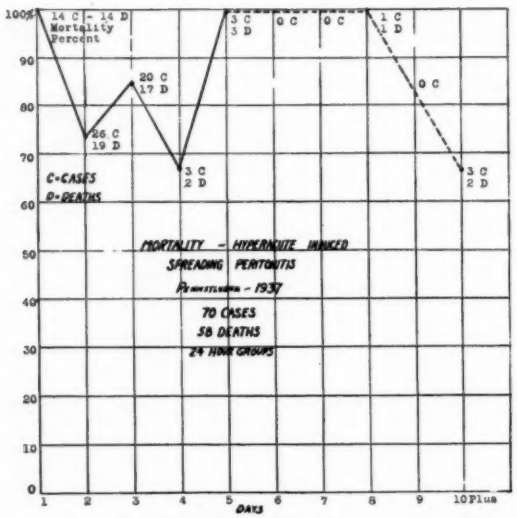
surgeons operating in Philadelphia, and 1,705 in Pennsylvania.

Table XXIV is shown to illustrate the improvement in management of this group of cases; the higher mortality in the State group is due in part to the greater number of catastrophes.

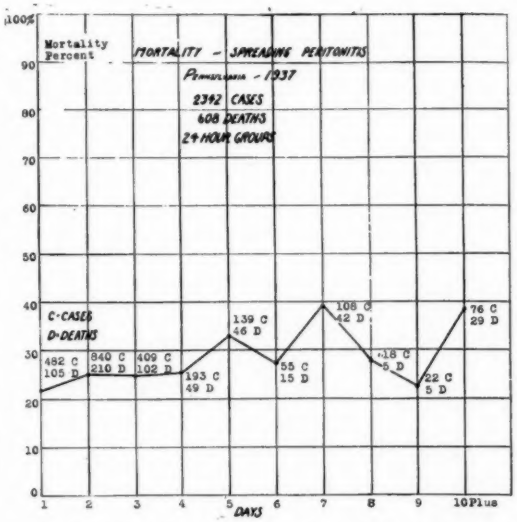
TABLE XXIV. APPENDICEAL ABSCESS

	Year	No. of Cases	No. of Deaths	Mortality Per Cent
Philadelphia	1928-29	1,502	57	3.79
	1930	625	11	1.76
	1931	618	10	1.62
	1932	572	5	.87
	1933	538	5	.93
	1937	458	3	.65
Pennsylvania	1937	1,080	13	1.20

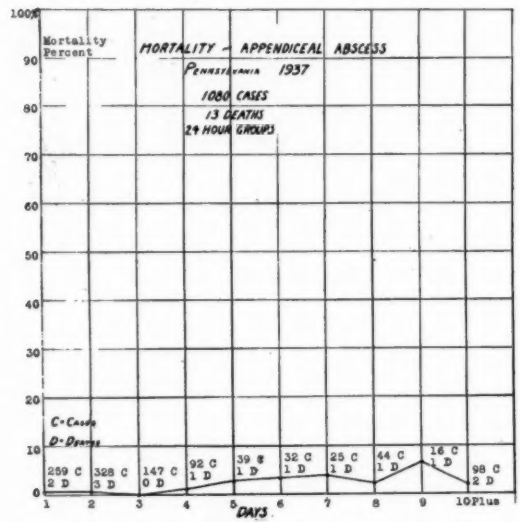
Table XXV contains data which summarize the active and potential factors responsible for the appendicitis-peritonitis deaths.



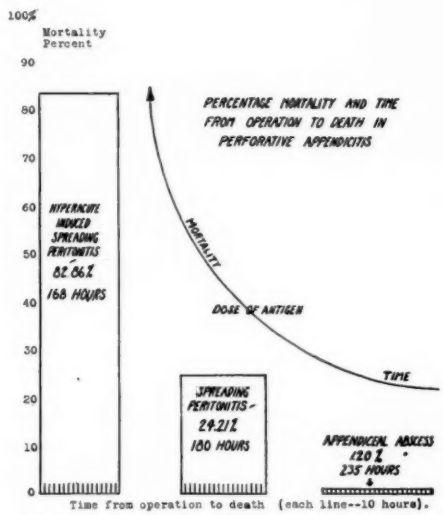
Graph 1.—*Hyperacute Spreading Peritonitis.* Before immunologic processes had developed, a large amount of antigen was projected into the peritoneal cavity—purulent distended appendices were ruptured at operation.



Graph 2—*Spreading Peritonitis.* Immunologic processes are in the early stages of development—operative procedures 83.73 hours after admission.



Graph 3—*Appendiceal Abscess.* Nature—patient's resistance—immunity is permitted to develop almost to its maximum—surgery is instituted after 123 hours.



Graph 4.

A summary of each of the four groups with comments has been presented. The problem at hand is the presentation of factors influencing the mortality which are common to all four groups. The writer is convinced that man is provided with but one system of defense against microörganismal invasion, that patients recover from spreading peritonitis following a perforated appendix in the same manner that they recover from a pneumonia or a typhoid fever. This is the premise on which the following rests. This conclusion was arrived at before it was realized that the answer to the query, "why do some patients live, and others die, of spreading peritonitis?"—probably awaits those who are interested in the science of immunology.

It has been shown by animal experimentation by many investigators, and it is demonstrated

TABLE XXV. APPENDICITIS-PERITONITIS

	TOTAL			AVERAGE				
	No. of Cases	No. of Deaths	Mortality Per Cent	Age	On Admission		Onset Symptoms to Oper. Hours	Operation to Death Hours
					Temp.	Pulse		
Hyperacute Induced Spreading Peritonitis	70	58	82.86	24	100	104	48	168
Spreading Peritonitis	1,118	269	24.06	22	101.4	118	69	144
Localizing Induced Spreading Peritonitis	1,154	281	24.35	30	99.7	100	98	216
Appendiceal Abscess	1,080	13	1.20	30	100.1	99	123	235

here, that absorption is increased in acute inflammation of the peritoneum. As the process diminishes in severity, absorption is diminished. *Time is an important factor.*

Microörganisms and their toxins vary markedly in virulence, but attenuation does occur as emphasized in the following: the highest mortality occurred in the group admitted within 48 hours (Graph 1), the second highest in those admitted within 83.73 hours (Graph 2), and the lowest occurred in the group admitted in an average time of 123 hours (Graph 3). Again, *time is an important factor.*

Time is also important in "what the surgeon does at operation." Is it necessary for him to hurry? The average time that has elapsed before admission to the hospital in the appendicitis-peritonitis group is forty-eight hours. The patient is one of the best surgical risks. Is it not best to make haste slowly?

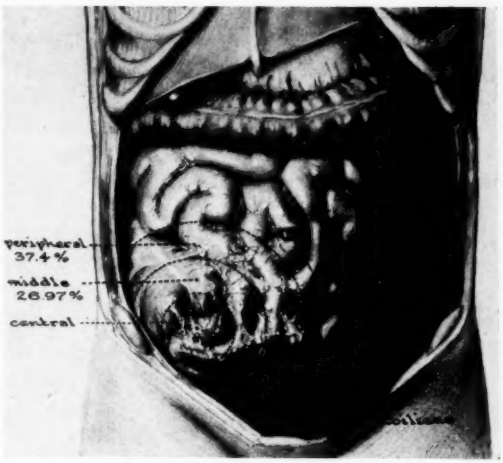


Fig. 4. *Spreading Process.* This is the spreading process. While the localizing process is regressing when operated upon, the spreading process is progressing. The mortality in this group is dependent upon the resistance of the patient, the time of operation, and what is done at operation.

DIAGNOSIS OF DISEASE OF THE CORONARY ARTERIES*

S. MARX WHITE, M.D., F.A.C.P.
Minneapolis, Minnesota

NO attempt is made here to consider systematically all the factors in determining whether or not the coronary arteries are diseased. While this is important, it is still more worth while to determine by examination the nature and circumstance of the disease condition as a whole, which is in truth the diagnosis.

Until or unless pathological changes in the coronary arteries cause impairment of the circulation in the myocardium, such pathologic changes are not recognizable during life. A single exception might be made in those rare cases where the fluoroscope or x-ray film have given evidence of calcification in the heart wall distinguishable from the calcification occuring in the aortic or mitral valve. Even this isolated type of demonstration is relatively insignificant because almost without exception when an atheromatous area is so far advanced as to give demonstrable areas of calcification, there is already impairment

*Read before the annual meeting of the Minnesota State Medical Association, Rochester, Minnesota, April 23, 1940.

of circulation in the myocardium beyond the point of calcification in the vessel wall.

For practical purposes then, it may be stated that the importance of coronary artery disease lies in its effect upon the myocardium and further that it is the effect upon the myocardium which we recognize, while the part played by the coronary arteries is largely a matter of inference. In other words, the true diagnosis from the clinical standpoint relates to the nature and circumstances of the disease condition in the heart muscle, and let us here include its conduction paths. We may infer the cause to lie in conditions in the blood vessels. Circumstantial evidence may make the inference valid.

It is necessary to consider, though with the utmost brevity, certain facts in the physiology and pathology of the coronary arteries, with particular attention to what is now known concerning the existence of anastomoses in these vessels and the possibilities in the development of collateral circulation.

The concept of Cohnheim, published in 1881, that the coronaries are end-arteries has been almost universally discarded. The work of Lewis Gross in 1921, of Spalteholz in 1924 and of Campbell in 1929 has been strikingly suggestive of the development of an increased anastomotic circulation with increasing age. Throughout all the discussions the suggestion of the importance of the Thebesian circulation has been heard, but it seems quite clear that this circulation is not of major importance, at least in the muscle areas which constitute the wall of the left ventricle and the interventricular septum.

It has been held for many years that the existence of anastomoses and the development of collateral circulation are the most important factors in the favorable response made by many individuals, even in the presence of more or less extensive, sometimes severe and acute, obstructions in the coronary arteries. It is probable that there is a wide difference in individuals in the extent of the anastomoses existing originally and in the capacity for the development of collateral circulation. The evidence of such suggested collateral circulation has not been readily secured. However, Blumgart, Schlesinger and Davis[1] have recently published the best planned study of collateral and anastomotic circulation in the heart to date. Their demonstration of extensive anastomoses in the heart with occlusions in coronary vessels is complete. In their conclusions they state that anastomotic circulation develops when and where it is needed, and that the development of such anastomoses is not related to age, for they are not present even in the hearts of senile patients when little or no coronary arteriosclerosis is present.

That obstruction in a coronary artery can cause damage up to and including actual tissue death, i.e., infarction, in the myocardium has long been known. In such a case this means, of course, that any collateral circulation which might be present at the time of the occlusion is inadequate. On the other hand, extensive and often multiple obstruction may be found without gross evidences of infarction, and the puzzle has been in the past to determine what underlies these extraordinary differences noted. The work of Blumgart and his associates goes far towards clarifying our concepts in this field.

In the management of the disorders dependent on disturbances in the coronary circulation the clinician has long been impressed with the part which time plays in the progress of the patient in whom evidences of myocardial damage have developed. Under favorable conditions of rest and with time and more time and still more time, each observer has seen cases of angina pectoris improve to the point of disappearance for a time at least, of the syndrome; has seen cases of myocardial infarction surviving the acute phase, return to more or less, and at times surprising, capacity; has seen remarkable improvement in the congestive failure group, though here as a rule the results are not so striking; and lastly may see sometimes favorable progress in those cases in which the chief expression of myocardial damage is through changes in conduction and in rate and rhythm of the heart.

For the clinician several problems present themselves. Such problems are:

1. Under what circumstances may the most effective collateral circulation develop?
2. How long a time is required for the maximum development?
3. How can the presence of effective collateral circulation be surmised or proved?
4. What methods of therapy and management, if any, will aid in the development of the desired adjustments?

These questions are not posed in order to ef-

fect an answer. When they are set before one in the study of a case they provide a stimulus for a more effective and useful appraisal.

The factors most important in the development of collateral circulation in the heart wall are as yet not clearly evident. Two factors, however, play the largest part.

The first of these is the individual variation. There is no opportunity to study this clinically and we can only surmise that it plays a part in the survival of one individual while another with an apparently similar lesion may succumb. The second factor is the element of time. It is important for us to study this factor because in the past we have relied upon it not knowing exactly why. Unfortunately we cannot see the processes of development of collateral circulation during life in the heart wall and can only estimate their presence by indirect evidence. A somewhat analogous situation, however, can be studied visually during life when there is an obstruction in the external iliac or the femoral vein on one side. Careful observation of the entire venous circuit visible over the upper thigh and lower abdomen in such cases shows venous dilatation in the collateral area very early, but the establishment of the maximum dilatation of the collateral veins and relief of the circulatory embarrassment takes many months and in some instances is not complete until two or three, and occasionally even more, years have elapsed.

Whether the development of collateral channels in the coronary arteries is more rapid or less so than in the venous circulation cited, one does not know as yet. It would not be surprising if the thicker, more resistant walls of arteries took longer for the development of the effective dilatations and anastomoses.

Until a sufficient series of well studied autopsy specimens can be secured in cases with authenticated dating of occlusion, we must rely upon clinical impressions. My tentative conclusion, based on the study of favorable progress in many cases of coronary occlusion and of angina pectoris, is that the full development of collateral circulation in the coronary arteries takes many months, a year or two and sometimes more.

After a myocardial infarction is demonstrated, it is seldom that an informed physician today will require less than two months, and will demand often more, before any activities whatever are allowed. If then symptoms or signs of circulatory embarrassment develop, it is usual that many more weeks and even months of relative quiet will be required.

Emphasis is placed upon the subject of the collateral circulation when considering diagnosis because it is the estimate of this collateral circulation and its effectiveness which plays a most important part in management. The diagnosis of the existence of circulatory inadequacy is not enough. An appraisal of the progress of compensation is essential. The realization is growing that this progress is in good part dependent upon compensatory collateral circulation.

There are four great groups of phenomena dependent on impairment of circulation in the myocardium and due to changes in the coronary arteries. These groups are: (1) The angina pectoris syndrome; (2) myocardial infarction; (3) myocardial damage with "congestive failure"; and (4) myocardial damage with changes in conduction and in rate and rhythm of the heart.

In many cases one or more groups of these phenomena are combined in the single individual. These combinations may be helpful in suggesting and in recognizing the vascular origin of the lesion. Time does not permit of a systematic study of the diagnostic features but a few remarks may be in order.

The angina pectoris syndrome when occurring without manifestations of any of the three other groups will at times present one of the most difficult diagnostic problems in medicine. This is particularly true when the economic problems of compensation and disability benefits are involved. The purely subjective character of the evidence in at least one-fourth of the cases will at times call for the greatest acumen and experience with even then the true answer not always immediately at hand. Some patients are unable to express themselves adequately and others may be so glib and well versed in a recital of typical symptoms as to arouse suspicion. Genuine differences of opinion between honest observers can thus develop. The credibility of a witness does not always impress itself alike upon all hearers. When to the subjective is added objective evidence, such as changes in heart size and in the electrocardiogram, the difficulties in diagnosis may disappear.

Although myocardial infarction has been receiving increasing attention since the publications

of J. B. Herrick beginning in 1912, it is still, I think, a condition which does not receive adequate attention by the majority of physicians. In my opinion the interests of the patient demand that whenever suggestive symptoms occur a careful continuing persistent study should be instituted promptly and this in satisfactory surroundings so that the question can be definitely answered: Is there in this case actual necrosis of tissue, i.e., definite myocardial infarction? To do that it is necessary to obtain evidence along all possible lines and to differentiate clearly from several conditions which may at times simulate it. It is not sufficient to acquire one or two lines of evidence but several must be studied, including a study for fever, leukocytosis, acceleration of sedimentation rate of red blood corpuscles, significant changes in blood pressure, signs of localized pericarditis or evidences of intraventricular or intra-auricular thrombosis, as shown at one time or another by embolism. Disturbances of rate and rhythm and the significant changes in the electrocardiogram are considered in another paper in this symposium. Later on in the case studies for the development of mural aneurysm with or without calcification are in order.

The importance of all this study in suspected myocardial infarction lies in this: That if a true infarct has developed and the patient survives the immediate event, a long, painstaking and studiously supervised period of rehabilitation is in order. The aim during this period of rehabilitation is to guide the patient with the greatest efficiency so that the proper combination of rest and time may be given to induce the development of the most effective collateral circulation possible. If it has been possible to stimulate a more intensive study of the angina pectoris syndrome and of myocardial infarction, the purpose of this paper will have been accomplished. The congestive failure group will be presented elsewhere and the electrocardiographic studies are considered by another participant in this symposium.

Reference

1. Blumgart, Herman L., Schlesinger, Monroe J., and Davis, David: Studies on the relation of the clinical manifestations of angina pectoris, coronary thrombosis, and myocardial infarction to the pathological findings. Am. Heart Jour., 19:1-91, (Jan.) 1940.

CORONARY DISEASE—ITS TREATMENT*

F. J. HIRSCHBOECK, M.D.

Duluth, Minnesota

THE "treatment of coronary disease" to the medical mind suggests the treatment of angina pectoris and coronary occlusion. For the sake of brevity and to limit the discussion to this most frequent phase of coronary disease, I shall not discuss the therapy of the various effects of coronary sclerosis on the myocardium of a more chronic nature, with or without heart failure. Naturally, some of the measures used in the treatment of the various manifestations of coronary disease overlap, but I shall restrict myself to the consideration of the treatment of angina pectoris and coronary occlusion.

Coronary Disease with Angina Pectoris

Angina pectoris is a functional state, and may be due to several causes, among them relative conditions of myocardial anoxemia due to paroxysmal tachycardia, hyperthyroidism or anemia; and due, in coronary sclerosis, to a diminished, quantitative supply.

Once the symptoms of angina pectoris are noted, certain therapeutic measures must be immediately instituted. If the anginal symptoms are in relation to an anemia, treatment must be directed to its cause. Thyroidectomy will relieve patients who have anginal symptoms in association with hyperthyroidism. Paroxysmal tachycardia related to hyperthyroidism can also be relieved by surgery. If due to other causes, quinidine is usually helpful. Syphilitic lesions affecting the coronary ostia in such a way as to diminish the blood supply to the myocardium must be treated with anti-syphilitic measures, preferably a milder non-reactivating agent.

Certain general measures are advisable in the treatment of angina pectoris with coronary sclerosis. Of these measures rest is by all means the

*From the Duluth Clinic, Duluth, Minnesota. Read at the annual meeting of the Minnesota State Medical Association, Rochester, Minnesota, April 23, 1940.

most important; restricted to bed rest, if pain is provoked on the slightest exertion or allowed to an extent and degree consistent with the patient's limited tolerance to exercise. Coöperation with the patient in eliciting the degree of tolerance usually enables one to arrive at a safe procedure. Patients may note how long a distance they may walk on the level before pain is induced, and how soon the pain response is developed by climbing stairs or walking uphill. It is emphasized to the patient that he must learn to keep his activities below the threshold which causes pain, and if pain comes on with any activity he must immediately discontinue such corresponding effort until pain no longer is initiated. As the condition of the patient appears to improve the amount of exercise indulged in may be prolonged gradually, until a fair or adequate compensation is arrived at.

Excitement and worry act similarly in inciting attacks, by accelerating the heart action and augmenting its work. The patient must be cautioned to avoid, when possible, situations which tend to excite him too much—quarrels, sad situations, watching or listening to the recital of athletic contests over the radio, etc. Many patients fail to recognize the influence of intercourse in affecting angina or coronary occlusive symptoms. Relinquishing a job because of anginal symptoms is a subject, however, that must be thought over very carefully before decided upon, because of the many personal economic and social factors which interplay. If the patient is overweight, weight reduction is undertaken. Since the patient cannot reduce by increasing his exercise, dietary management is oftentimes our only recourse. Weight reduction should be accomplished gradually and consistently rather than to enforce too marked a nutritional deficiency with the hazards of an avitaminosis added.

The consensus seems to be that tobacco should be avoided, and its indictment seems to be founded on a clinical and experimental basis. Compromising with the patient to reduce the amount of smoking is usually ineffective, and complete abstention should be insisted upon. Alcohol in medicinal doses apparently is of benefit in promoting vasodilatation, but should be used in medicinal proportions and not in excessively stimulating amounts. Kerr has suggested the use of a belt for support of the abdomen, so as to facilitate diaphragmatic function and respiratory efficiency. In a very limited experience its use has been satisfactory, and apparently pleasing to the patient.

Medicinal Treatment.—Whiskey has already been mentioned as of value. The nitrites have been found most useful in relieving attacks of angina pectoris, and according to several clinicians, have also been used to prevent attacks with evident success, administered just before effort. Nitroglycerine tablets dissolved under the tongue or crunched between the teeth, and thoroughly dissolved, in doses of 1/100 gr., seem to be the most effective method of use. Amyl nitrite is satisfactory, but less easy to handle, and because of its odor, conspicuous in its use. Recently, inhalers containing octyl nitrite have been advised as beneficial. The routine use of the nitrites is of doubtful value.

The xanthine derivatives have been widely used in routine practice, and good results reported in a large percentage of cases. There is, by no means, universal agreement among writers, however, as to the relative merits of this group or various members of the group, some preferring theobromine compounds, others theophylline preparations. These drugs may be used for a long period of time unattended by any ill effects. I believe there is a tendency to use xanthine derivatives in too small doses. Recently, Clerc and Sterne[1] drew attention to a group of substances influencing anginal crises, which unite the antifibrillatory properties of quinic substances, and the beneficial effect of theophylline on the coronaries. It is known as 1262-F, the diethylaminoethoxydiphenyl, and is administered by mouth, up to daily amounts of 0.15 to 0.2 gms.

Many patients with anginal symptoms are nervous and high-strung, and even in the more calm, mild sedatives are frequently advantageous. For the simple nervous situation, bromides or the barbiturates are sufficient. With more active pain, codeine may be used. In 1939, Master, Jaffe and Dack[2] made a study of sixteen drugs, including a placebo, namely, milk sugar, on angina pectoris, to note evidences of any specific effect on the anginal syndrome. They included several xanthine derivatives, alcohol, sedatives such as phenobarbital, chloral and bromide, the nitrites, tissue extracts, digitalis, and two narcotics, codeine and dilaudid. Some patients were helped by all the drugs; others by none. No

drug was consistently successful in a significant number of cases, and equally satisfactory results were obtained with the use of the placebo as with the supposedly more active drugs. The writers believe that much of the benefit is derived from psychologic factors and a reduction in the emotional status. Their observations do not apply, of course, to the acute attacks. Long clinical experience, I think, tends to support the view of these writers, at least, in part. The value of routine medication must not be over-estimated.

I believe there is little basis from the standpoint of clinical results as far as the use of tissue extracts is concerned. In certain instances the exhibition of sedatives, xanthine derivatives, nitrites or alcohol seems beneficial; in others not. The choice of the agent used and the duration of its administration must depend upon the personal desires and choice of the physician. Personally, I am inclined to the use of small doses of sedatives, with one of the xanthine derivatives, and frequently, alcohol in proper doses.

In the hands of a few, the surgical treatment of angina pectoris has been highly extolled. On experimental evidence, and occasionally in humans, good results are reported by such men as Beck, O'Shaughnessy, and others. The basis of the procedure is an effort to promote collateral circulation to the myocardium by the use of adjacent normal tissue, such as muscle or the omentum. Such procedures are not within the province of this discussion, and not recommended at this time for general practice. Thyroidectomy, as first advised by Levine, is apparently justifiably falling into disfavor. In some instances, where the patient's anginal symptoms are frequent and difficult to relieve, even with a most careful regimen, peravertebral alcohol injections may be used. They are not unattended with danger, and should be used only by the experienced, and with due caution. In ideal circumstances, the results are good, but it is to be borne in mind that though the pain discomfort may be diminished, the pathologic process, which is the basis of the disease, is not altered.

Treatment of Coronary Occlusion

The first consideration in the treatment of patients with coronary occlusion is the relief of pain and shock. The latter varies considerably in its degree, but one can never tell when the physiologic mechanism of the heart may be interrupted and death result, even in the absence of shock. Hence, rest (using the term in a broad sense) is an imperative therapeutic necessity. If the patient's condition is critical it may be injudicious to move him, unless the attack comes on in the street or in a shop. Oftentimes, it is best not to disturb him by undressing him except for his outer clothing. If the patient breathes more easily in the semi-upright position the body should be adequately supported by pillows. Effort should be made to keep the body warm. Occasionally in syncopal attacks the head may be kept flat. No one should be allowed in the room except the immediate attendants. The alarmed relatives should be in the background. These measures suffice for the immediate situation. Morphine is the best remedy for the relief of the excruciating, wearing-down pain. In vigorous persons ½ gr. morphine sulfate by hypodermic, or ¼ gr. by the venous route is not excessive, even with due regard to certain unfavorable after-effects, as for example, vomiting and respiratory depression. Vomiting is so commonly associated with severe attacks that one is not justified in always blaming morphine for its occurrence. I see no great advantage in substitutes, such as dilaudid or pantopon. If the patient's resistance to pain seems to be good, or the attacks associated with less pain, smaller doses of morphine may be sufficient. It is my belief, however, that pain, combined with shock, is so distressing to the patient that its relief should be occasioned as rapidly as is consistent with safety. Atropine and/or paparetine may be tried first or as accessories to morphine.

There is also evidence, as pointed out by Eppinger, that under the influence of morphine a smaller cardiac output is required, and the resultant diminution of work on the part of the heart may be of importance. Nitrites are of no value in acute coronary occlusion. Most physicians are inclined to give an initial dose in the hope that it may relieve what might be a simple anginal attack. An additional agent of undoubted benefit in the relief of the immediate attack when other measures fail, is the inhalation of oxygen in adequate percentage amounts. It is indicated in all cases where there is an increase in the pulse rate, respiratory rate, dyspnea, or cyanosis. Oxygen relieves all of these if the condition is not irreversible. It also tends to lessen pain, and is an adjuvant to the effect of morphine. Concentra-

tion should be at about 50 per cent, and because of the ease of its administration and the lack of physical annoyance, an oxygen tent is the better method, though the nasal catheter is of great benefit.

It is not necessary to give the patient any food for the first few hours after the attack, but fluids may be administered by mouth, preferably slowly, and with due regard to the likelihood of vomiting. Water (cold, if desired, or hot if the patient is in shock) in frequent small amounts is usually sufficient until the patient recovers from the initial shock. After this occurs it may be increased in amount to an optimal level, or even increased to 2 or 3 liters per day if the patient perspires excessively. If vomiting continues, physiological salt solution, with isotonic glucose, may be given subcutaneously slowly. This is much more satisfactory than the rectal drip, and less likely to be harmful at this stage than intravenous medication. The use of hypertonic glucose solution in one form or another (50 c.c. in 50 per cent solution, one to three times per day) is advised by some clinicians, on the basis of improving the myocardial nutrition. Theoretically, it might be considered of advantage, but I should consider an isotonic subcutaneous administration sufficient for the purpose if the patient is not able to take sugar by mouth, in the form of fruit juices, to which sugar may be added. In patients with pulmonary edema or passive congestion, I am inclined to its use in higher concentration. Because of the dehydrating effect of concentrated glucose solutions on the tissues, adequate water intake must be supplemented.

The function of the bowels may be maintained by giving an enema in from twenty-four to seventy-two hours, depending, on the one hand, upon the degree of shock, and on the other, the presence of abdominal distention, which may be relieved by such measures. Individual circumstances must help the clinician in making a decision, but insistance on having the bowels move once a day is, I believe, in most cases unnecessary. After the first few hours, food, in liquid form, may be administered, to be followed by semi-liquid preparations, such as cooked cereals, light puddings, pureed vegetables, soft eggs, et cetera, and later supplemented by articles of food which can be prepared by being well cooked and yet pleasing to the patient's palate. Just as it is important not to disturb the patient too much physically by getting him on the bed pan frequently to permit of bowel function, it is equally important not to examine the patient too frequently by turning him over on his side or by raising him up in bed. There is no condition in which greater caution must be exercised in needlessly moving the patient about for the desire of a thorough examination of the lungs posteriorly, than in coronary occlusion.

After the treatment of the initial shock and the insistence upon absolute rest, the question of further safeguarding the patient's physical capacity and stamina must be considered. The question of bed rest requirements as far as duration of time is concerned is frequently brought up by the patient himself as he begins to improve. I do not believe that any patient should be allowed to get up in less than four weeks after a coronary occlusion, and oftentimes he must be kept in bed for a much longer period of time, depending upon various contingencies. A period of six weeks is the average. One is guided by certain clinical laboratory findings—the white blood count, differential blood count, and the sedimentation rate. The temperature curve and pulse rate and the character of the physical findings are also criteria for evaluation. No patient should be allowed to get up for one week after all evidences of activity, as manifested in one way or another, subside.

When the patient is allowed to get up it must be done in very gradual increments of time. It must be remembered that reclining in a chair is frequently no more burdensome than lying in bed, and probably less so. The hazard lies more particularly in the effort to transport him between the bed and the chair. From the time the patient takes his first step, the time and distance involved in exercise may gradually be increased, with small excursions about the room, to the bathroom (on the same floor), and as the patient improves, gradual descent and ascent of the stairs. In people of small stature and weight it frequently helps the morale considerably to carry the patient downstairs and up for a change of milieu. It is said that descending the stairs involves five times the physical exertion encountered in walking the same distance on the level, and ascending the stairs, fifteen times as much effort. This may be used as a rough criterion for an increase in effort. As soon as the patient is able to do so, short walks out of doors, or rid-

ing about in an automobile may be indulged in, future increases dependent upon the response to each added effort. It is needless to go into greater detail. The resumption of the patient's usual activities must also be gradual. When possible, a sojourn in warmer climes in the wintertime is of benefit, because of the lack of necessity of combating the severe element of climatic change. The patient should be safeguarded first in going outdoors in inclement weather, and the necessity for donning heavy clothing to combat the winter atmosphere should be avoided.

Medicinally, the patient may be given sedatives if his rest is disturbed, or if he is unduly apprehensive about his condition or his future. The barbiturates are effective, with no contraindication. Routine medication with the xanthine derivatives is a moot point, but is used by most clinicians for a few weeks to a few months, in the hope that it may increase the circulatory efficiency generally, as well as in the myocardium. Theophylline derivatives may be used in smaller doses, but as compared to the theobromine derivatives probably have no superiority, and may, in fact, be somewhat less efficient, when given by mouth. The use of quinidine as a preventive of ventricular fibrillation or other physiologic disasters may be helpful, but convincing evidence is lacking. If the patient presents premature ventricular contractions the indication is more definite. Three to 5 grains may be repeated three times a day.

The consensus is that digitalis should not be used except where indicated for reasons consistent with cardiotherapeutics in other conditions. If there is a fibrillation present and the rate increased, nothing is so effective and if the patient exhibits signs of stasis in the lungs or general circulatory insufficiency it may be used according to the same method as employed in heart disease due to other causes. When a threshold digitalization has been attained the dosage should be reduced to 1 or 1½ cat units per day.

Certain situations may arise as complications in coronary disease which demand other measures. In patients who are in shock and in whom the blood pressure is low, adrenalin may be of benefit. There may be some objection from the standpoint of physiologic experimentation, but clinically, in discussing the matter with other cardiologists, they admit of certain clinical enthusiasm in regard to it. If the patient has pulmonary edema during or after the attack of occlusion, morphine is necessary; adrenalin may be used as well as aminophylline in 7½ gr. doses intravenously once or twice a day. Concentrated glucose preparations one to three times per day are indicated. Measures for the promotion of absolute rest must again be enjoined, in the event of such attacks occurring sometime after the actual occlusion.

In the patient with congestive heart failure digitalis is the sovereign remedy, combined with a reduction in the fluid intake, at the same time observing a record of the intake and output, and the use of diuretics. It is in these instances that the xanthine derivatives, given by mouth, are of benefit, and less disturbing to the digestive apparatus than corresponding large doses of the ammonium salts, etc. Mercupurin, salyrgan, esidrone, or any similar preparations, with or without theophylline, intravenously, two or three times a week, are of great benefit in relieving the patient, not only of the mechanical distress of the congestion, but also in yielding physiological relief. In patients who are in shock, the question of giving stimulants, such as coramine, caffeine, or strychnine, usually lies within the choice of the physician. Except as a gesture, I have relatively little faith in them.

I think there are a few points in the treatment of coronary occlusion that are needful of particular emphasis; namely, the insistence upon absolute physical and mental rest, and the sufficient administration of morphine and atropine and possibly oxygen in the early phases of the attacks; the need of keeping the patient quiet long enough so as to permit of anatomic restitution, at least in part, to the injured structure; and the avoidance of over-treatment of the patient by needless medical and nursing manipulation. Generally speaking, the patient, after the initial attack has been treated and overcome, should be permitted to coast along in his convalescence, until he is permitted to resume his activities gradually.

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THE ELECTROCARDIOGRAM IN CORONARY DISEASE*

HARRY OERTING, M.D.
Saint Paul, Minnesota

IN considering the electrocardiogram in coronary disease, I will not present lantern slides of typical and atypical tracings, and I will not explain the technicalities of the electrocardiograph itself, but limit my discussion with you to what you can expect to learn from an electrocardiographic tracing and what the limitations of this procedure may be.

In discussing this subject it is necessary that we first state briefly the fundamental facts regarding the electrocardiograph. The electrocardiograph is an instrument primarily concerned with furnishing evidence of the anatomical state of the heart muscle and of the coronary arteries, and in no way does it aid one in determining the functional capacity of the heart or its ability to do work. In the very small proportion of cases of cardiac irregularity, that cannot be differentiated clinically, the electrocardiograph can clarify the exact nature of such an irregularity. The electrocardiograph has almost no value in diagnosing valvular lesions.

The typical case of coronary occlusion can be diagnosed from the clinical picture alone and the electrocardiogram is needed only to localize the site of the infarction. In the atypical or questionable cases an electrocardiogram will often definitely decide the matter immediately, although at times when the clinical history and findings are inconclusive, serial records may be necessary to rule out or establish the diagnosis. It is imperative always to bear in mind the fact that occasionally all records may be within normal limits and yet the patient dies of coronary occlusion. This is usually in rapidly fatal cases where cardiographic changes do not have time to manifest themselves. It is well to reëmphasize that serial records are not only very important in the diagnosis of coronary occlusion, but they also give a graphic picture of the course of the disease and thus make the most efficient management possible.

In the arteriosclerotic age group, serial electrocardiograms give an important picture of the ageing process in the coronary blood vessels and may indicate changes long before there is any clinical evidence. Therefore, in systemic diseases in which heart disease of an arteriosclerotic nature is common, it is prudent to make periodic electrocardiographic records. The electrocardiogram correctly correlated with the clinical history and physical findings, materially aids in judging the progress of coronary disease.

Substernal pain and distress is one of the most serious problems confronting the private practitioner and the entire mode of life, economic position and happiness of the patient and his family depends upon a correct diagnosis. As we all know, chest pain is not always due to coronary disease or coronary insufficiency, and the electrocardiogram will often assist us in arriving at a diagnosis. Unfortunately electrocardiographic records are not always diagnostic. We all know that one can have organic heart disease with the pain due to other causes and that chest pain of cardiac origin does occur in some cases even in the absence of electrocardiographic abnormalities. Successive changes in the serial electrocardiogram is the most important diagnostic aid of electrocardiography and often the electrocardiograph is able to eliminate the heart as the source of symptoms suggestive of myocardial disturbance.

The electrocardiograph is a most useful instrument but routine electrocardiograms in the absence of clinical findings of heart disease, certainly are of no value to the clinician in arriving at a judgment of the condition of the heart.

All electrocardiographic records must be correlated with the clinical history and physical findings and the electrocardiogram of and by itself is not able to make a diagnosis or a prognosis for you. Owning and using an electrocardiograph involves more than just making a record—a twelve-year-old can do that after fifteen minutes' instruction. You must be willing to undertake sufficient study to enable you to make an intelligent interpretation of the records you secure and properly correlate them with the clinical findings, or in lieu of this, your records must be interpreted by a qualified person and in order that this

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interpretation may be of the utmost value, the interpreter must have a summary of the clinical history and physical findings.

Summary

1. The electrocardiograph definitely aids in the diagnosis and management of coronary disease and serial electrocardiograms are invaluable.

2. The electrocardiograph is only informative on the anatomical condition of the heart and in no way aids one in determining the functional capacity of the heart or its ability to do work and it is of almost no value in the diagnosis of valvular defects.

3. The electrocardiograph may eliminate the heart as a source of symptoms suggestive of myocardial disturbance.

4. The interpretation of the electrocardiogram must be made by a thoroughly trained individual and the findings must be correlated with the clinical history and physical findings.

5. The electrocardiograph, of and by itself, cannot make the diagnosis and prognosis for you.

PNEUMOCONIOSIS*

A. J. LANZA, M.D.
Assistant Medical Director
Metropolitan Life Insurance Company
New York, N. Y.

PNEUMOCONIOSIS is a generic term whose use at times has been attended with some confusion. It embraces the reactions of pulmonary tissue to industrial dusts, which in some instances present clear-cut diseased conditions; in others, conditions which are not entirely clear; and in still others, conditions which have little or no clinical significance. Much water has gone over the dam since attention was first called to the importance of dust exposure in industry as a cause of pulmonary disease. A little over thirty years ago, when the campaign against tuberculosis was well under way, the statisticians began to call attention to the fact that workmen in dusty trades had a consistently high death rate from tuberculosis. It was surmised that many kinds of dust, by virtue of their physical properties of hardness, sharpness, and insolubility, produced a mechanical injury of the lungs which paved the way for a tubercle infection. With respect to certain organic dusts which did not have physical properties calculated to injure the pulmonary tissue, it was felt that these probably acted as conveyors of the tubercle bacillus. This seemed a reasonable explanation for the excess mortality from tuberculosis observed among certain groups of textile workers, tobacco workers and leather workers.

Disability and a high mortality were so conspicuous in what was commonly termed the hard rock mining industry that it was but natural that anti-tuberculosis workers should stimulate investigation of that type of underground work and the first clinical study of pulmonary dust disease was made about twenty-five years ago by the United States Public Health Service and the Bureau of Mines, to be quickly followed by others in various parts of the country. These early investigations were mostly confined to the mining industry, except for the studies made in the textile industry by a group of physicians in Philadelphia.

Investigations were made in industrial communities over long periods of time. This procedure offered the tremendous advantage of observation of industrial workers ranging from those new in the industry to those who had been employed for many years, and thus made possible a correlation between the symptoms they presented and the length of employment and the working conditions in various occupations in the same industry. There was gradually built up a comprehensive knowledge of the etiology of pulmonary dust disease which led to a clearer understanding and did away with many false preconceptions. The most conspicuous fact was that where workmen were exposed to dust containing free silica in large amounts, they contracted a definite and specific type of pulmonary fibrosis; that this fibrosis produced characteristic appearances on an x-ray film, and was accompanied by a consistent progressive clinical picture, in which dyspnea on exertion was the outstanding symptom.

It was learned in time that the symptoms pre-

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sented by any group of workmen bore a definite relationship to the severity of such silica exposure. In other words, the symptoms and progress of the disease were directly proportionate to the amount of silica which invaded the pulmonary tissue. It was conspicuously apparent that in communities where men were exposed to a severe silica hazard, the incidence of and mortality from tuberculosis was extremely high, in some instances so high as to be hardly credible. This tuberculosis mortality, however, was in sharp contrast with that of surrounding communities and even with that of the workers' families.

There was built up a concept of an occupational disease resulting from the inhalation of free silica dust, manifested primarily as a fibrosis evenly distributed throughout both lungs, which, dependent upon the circumstances of exposure to silica dust, would progress with the gradual development of disability. Secondarily, especially where the silica damage to the lungs was of a considerable extent, tuberculous infection was apt to appear. Infection with the tubercle bacillus was more than merely a terminal episode and these two conditions reacted upon each other, definitely increased disability, and frequently led to a fatal termination. When death occurred as a result of pulmonary involvement in the great majority of cases, infection with the tubercle bacillus was present.

There came to my attention within the past month the report of the Miners' Phthisis Prevention Committee of Johannesburg, South Africa, for the three-year period 1935-1938. The authors summed up the experience of thirty years' study of silicosis in the gold mining industry and it was interesting to note how largely the observations concerning the etiology and clinical course of silicosis made in the early studies in this country had stood the test of time. Laboratory researches which have been carried on in this and other countries have modified our earlier ideas as to the manner in which silica affects the lung and have greatly increased our knowledge of the pathology of dust disease. Modern roentgenology has made possible early and accurate diagnosis and also has enabled the physician to watch the progress of affected individuals.

I might state here that organic dust has not been shown to produce a reaction of the pulmonary tissue and the pronouncement of Landis some twenty years ago that organic dusts played no part in causing pulmonary fibrosis has held good.

Attention was naturally directed to other industries which involved exposure to dust containing silica in many forms—that is to say, combined silica—as well as exposure to other types of non-siliceous inorganic dust. Here investigators found that the picture of disability and disease seen among those exposed to free silica was conspicuously absent. Various terms such as chalicosis and siderosis fell into the discard and other designations, such as potters' rot, grinders' rot, miners' phthisis, and miners' consumption were found to relate to identical conditions and were supplanted by the term silicosis.

Field studies of various industries went hand in hand with animal experimentation in which were employed dusts similar to those found in industry. Animals were exposed to the inhalation of these dusts in specially prepared dusting chambers and Gardner, at the Saranac Laboratory, was the first to demonstrate silicosis in experimental animals. The method of intraperitoneal injection, with the technique of Miller and Sayers, was also used. The underlying motive for all this was to determine which industrial dusts were harmful, that is, would cause a pneumoconiosis of such a type or degree as to produce disability.

Among the dusts investigated both in industry and in the experimental laboratory were limestone, calcium carbonate, gypsum, cement, feldspar, soapstone, talc, precipitator ash, artificial abrasives, sericite and others. None of these, whether they contained silica in combined form or no silica, produced disease in animals, nor was evidence of disease found among workmen exposed to these dusts except in those instances where free silica was also present.* That the failure to find evidence of pulmonary disease among workmen was not due merely to lack of a sufficiently severe exposure was indicated by the uniformly negative results in animals where the conditions of exposure were known and controlled and similar to those of silica exposures. X-ray films of individuals exposed for a number of years to these inorganic dusts, which contained no free silica, may show peribronchial thickening or accentuation of the bronchial tree. The same appearance can be found in individuals who have never been employed in dusty industries. Occa-

*Talc is one exception. See U.S.P.H.S. study 1935.

sionally x-ray films of some of these workers will show a diffused haziness or increased density that might be construed as due to the action of dust. However, these workmen are healthy and neither symptoms nor disability can be ascribed to this x-ray appearance. Hence, my opening comment that pneumoconiosis, as a generic term, has a limited importance from a clinical standpoint.

Two specific and distinct types of pneumoconiosis are important, namely silicosis and asbestosis. Asbestosis is the exception to the general rule in that it is a silicate, a form of combined and not free silica, which does produce a characteristic fibrous reaction in the pulmonary tissue and which may produce disability and death. In animal experimentation, intraperitoneal injection of asbestos dust produces an inert reaction—that is, there is no cellular proliferation as is observed with the various forms of silica. Inhalation experiments produce a pulmonary fibrosis similar to that in human beings, with the curious anomaly that fibrosis did not result when the finer dusts were used. This has led Gardner to believe that the action of asbestos in the lungs is probably mechanical and due to the peculiar shape of the asbestos spicule and the constant movement of the lungs. Further support is given to this theory in that, unlike silica, asbestos will not produce its characteristic reaction in animals in other than the pulmonary tissue.

Asbestosis first came into prominence in this country about 1930, since when a number of industrial studies have been made. The symptoms are largely similar to those of silicosis but the pathology and the appearance on an x-ray film are entirely different. The x-ray shows a diffuse haziness or ground glass appearance, especially in the lower half of the lungs; nodulation is absent, and the factor of infection appears to be much less important than in silicosis, at least that is our experience in this country. The total number of workmen exposed to asbestos dust inhalation is very much smaller than for silica and strenuous efforts have been made by asbestos plants to control their dust hazard. Asbestos fabrication is a factory industry, largely textile in character, and the control of dust is less difficult than the control of silica dust in mines and foundries. Consequently, there has not been and probably never will be, the opportunities for the clinical study of asbestosis such as have been made possible by the wide variety and extent of silica occupations.

A small number of deaths from asbestosis have been reported and not all of these have been followed by postmortem. Mostly other organic disease was also found or diagnosed, which tends to confuse the rôle of asbestos in these cases, but in some cases death undoubtedly resulted from uncomplicated asbestosis.

There has not been sufficient opportunity in field surveys of asbestos plants to acquire enough information to correlate the production of asbestosis with varying degrees or intensities of dust exposure nor have the conditions of dustiness, as evidenced by particle counts, run as high as in the more severe types of silica dust exposure. We have had under observation for ten years a small group of workmen with asbestosis. Some of these have shown a progression on their x-ray films. They are still working and in seeming good health. However, in the meantime the plant has been cleaned up and the dust controlled so we do not know what would have been their history had they continued to work under the former, presumably hazardous conditions.

Our original concept of silicosis was based on studies of industries in which there was generally a more or less severe exposure to dust containing a high percentage of free silica. As industrial investigations came to include a wider variety of industries, our concept broadened and underwent some modifications. The data gained in industrial surveys, when correlated with laboratory findings, cleared up a number of apparently confiicting and puzzling observations. Not only did we learn that the incidence and progress of silicosis in any location depended upon the amount of silica dust in the air and the length of exposure, but it was demonstrated that in many instances other substances were present in the dust with the silica and that these substances modified the action of silica. Furthermore, it was shown that the action of silica was not due to its physical properties but to its chemical properties. Also, where certain substances had been accused of causing a pneumoconiosis, the offending agent was in fact the silica which was also present. Metallic dust, for instance, which in the early writings had been considered as more dagerous than silica, was found to be relatively harmless. There came a better understanding of what caused the variations in the clinical appearance

and course of silicosis as observed in different locations and in different industries. Men engaged in iron ore mining and in quarrying and cutting granite are exposed to dust containing silica and other substances as well, which tend to retard the action of silica upon the lung tissue. The pathologic process is altered and the appearance on the roentgenogram is altered sufficiently for the experienced physician to distinguish one type of exposure from another. With the mixed dusts, the progress of silicosis is usually slower and when infection occurs, it tends to be more chronic but the amount of tuberculosis found among granite workers and iron ore miners is nevertheless inordinately high. Cases have been reported of workmen exposed to dust containing silica and caustic alkalies which produced a rapidly developing and fatal type of silicosis with early infection.

On the other hand, certain industries have been studied in which the silica hazard was found to be of a degree which would not produce disabling silicosis within the average working lifetime. After forty or more years of such exposure, many workmen would show typical nodulation on the roentgenogram, but their symptoms were few or absent and they were still working every day, many of them being well over sixty years old. All of this emphasizes how necessary it is to study the etiological factors in order to arrive at a correct solution of the silicosis hazard in a given location or in a particular industry. It also explains the apparently conflicting conclusions that are sometimes drawn from isolated cases of silicosis when the work conditions, that is, the nature and type of exposure, have not been or cannot be determined.

The pneumoconiosis of coal miners illustrates very well the modifying action of one dust upon another. Miners' asthma had for years been recognized by physicians in the anthracite industry as a definite clinical entity. It was difficult to believe, however, that coal, an organic substance, could produce a disabling pneumoconiosis. The Public Health Service made a thorough study of the anthracite industry in 1933 and the results of this confirmed what many observers had suspected, namely, that disabling miners' asthma was, in fact, a silicosis; a form of silicosis modified by the combined action of silica and anthracite dust, and to which the term anthraco-silicosis might justly be applied. The incidence and progress of disability was, as in other silica dust hazards, proportionate to the extent of dust exposure. The silica was accounted for by the fact that the veins of coal lie between rock formations which frequently have a fairly high silica content, and, also, that there was some silica in direct association with the coal itself. Consequently, the workers had some silica exposure. Anthracite mine dust varied in its silica content from about 11 per cent for miners working directly in coal to about 60 per cent for men working in rock, with various types of exposures falling between these two extremes. Pulmonary tuberculosis was associated with anthraco-silicosis to a very considerable extent.

Below the age of thirty-five, hard coal miners showed an incidence of pulmonary tuberculosis slightly less than that found among male workers in general. In the age group of thirty-five to forty-four, the tuberculosis incidence was twice as great, and from forty-five to fifty-four, five times and above fifty-five about ten times the rate for all adult male workers. Or, to express it in another way, clinical pulmonary tuberculosis was found in 15 per cent of those with early anthraco-silicosis and in 43 per cent of those in the more advanced stages. The x-ray appearances, while similar to the silicosis of hard rock miners, can usually be differentiated from the latter.

With respect to bituminous coal mining, the silica factor is apparently less in extent or lacking entirely. A number of studies have been made by the Public Health Service but have not yet been published. Death certificates of soft coal miners were analyzed for a five-year period and showed no appreciable difference, with respect to deaths from tuberculosis, between the coal miners and the farmers living in the same counties.

Inhalation experiments with bituminous and anthracite coal (1.76 per cent SiO_2) have produced a very slight reaction and anthracite dust proved inert on intraperitoneal injection. We may conclude that in the absence of silica, coal will not produce a specific fibrosis associated with disability.

The relationship between pneumonia and silicosis is not entirely clear. The data obtained from various studies and from animal experimentation are not always consistent. The high incidence of pneumonia in some industries with a silica hazard can not be disassociated from ex-

posure to extremes of heat or cold or both. Coal miners have a high incidence of pneumonia and it is a common cause of death among the older men with anthraco-silicosis. My own impression is that individuals with any considerable amount of silicosis have a more unfavorable prognosis in pneumonia than would otherwise be expected but this experience is not very recent. It remains to be seen whether silicotics respond to specific serum treatment or chemotherapy as well as do those who do not have dust fibrosed lungs.

Summary

There is still much to learn about the action on the lungs of many kinds of dust. The effects of pure silica (SiO_2) have been demonstrated under many conditions in this and other countries with a high unanimity of agreement among investigators. Where the silica damage to the lungs is extensive, the incidence of tubercle infection is high. Where the silica exposure has been extreme, a tuberculous infection is almost inevitable, but every physician who has had wide experience with these cases has seen exceptions to this rule.

Today in American industry, severe exposures are uncommon. They occur mostly as isolated instances as when a sandblaster has been working with faulty protection. Tuberculo-silicosis may run a very chronic course, but if the silica damage is extensive, the progress may be fairly rapid, but many exceptions will be found to the general rule.

We are acquiring a constantly broadened knowledge of the action of mixed dusts, that is, dusts containing other substances along with the silica, but the more intense the silica exposure, the more these cases run true to type. Naturally the clinical picture of silicosis is changing as industrial dust control becomes more and more perfected so that we may expect to see more and more of the type or degree of silicosis which is accompanied by little disability. I am convinced, however, that where the reaction has proceeded to cause nodulation, even though moderate in extent, a definite, increased susceptibility to tuberculosis takes place.

Finally, we must consider the criteria upon which the diagnosis of silicosis can be based. Undoubtedly, the early manifestation of silica dust reaction is peribronchial thickening with an increase of the hilium shadows, as seen in a roentgenogram. The same may be seen in workmen exposed to dusts other than silica and in individuals who have never had any sort of dust exposure. Consequently, those of us who got together several years ago under the leadership of Dr. Pancoast, felt that for practical purposes, a diagnosis of silicosis was not justified unless the specific reaction of nodulation, as found in experimental animals as well as in human subjects, was present.

In addition to a characteristic film, there must be a history of exposure to silica dust adequate to produce disease. Where an industrial investigation is being made, and the working conditions previously determined, it is usually not difficult to separate the silicotics from the non-silicotics and usually the presence of infection can be recognized or ruled out, careful consideration being given to the physical signs and the symptoms as well as the x-ray film. The isolated case that may turn up in hospital or clinic service may present considerable difficulty. Such patients are apt to be suffering from a combination of illnesses. They usually cannot give a sufficiently informative work history and even with a characteristic film, it may be difficult to say that a patient has silicosis (or even more difficult, asbestosis) and to what extent his pulmonary condition may be responsible for his symptoms or his disability.

INFECTED DERMOID CYST OF THE THORAX SIMULATING CHRONIC EMPYEMA*

HOWARD K. GRAY, M.D. and ROBERT WOODRUFF, M.D.

Rochester, Minnesota

AN INFECTED dermoid cyst of the thorax which simulates chronic empyema is interesting from two standpoints: first, because of the rarity of this manifestation of a dermoid cyst, and, second, because of the difficulty of diagnosis.

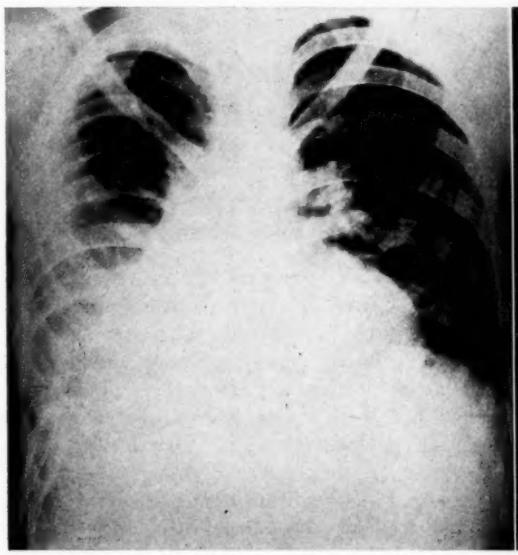

Fig. 1. The thorax prior to operation. The increased density in the lower lateral and posterior portions of the right side of the chest was interpreted as being due to markedly thickened pleura, probably associated with chronic empyema.

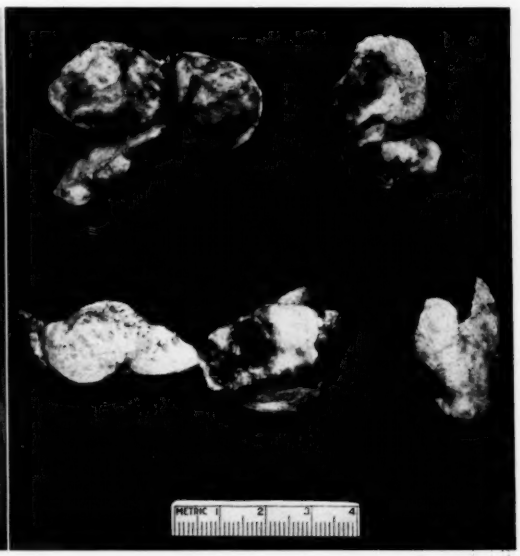

Fig. 2. Pedunculated growths removed at operation. The epithelial nature of the covering can be noted and a few fine hairs can be seen protruding from the tumors.

Dermoid cysts of the thorax arise practically without exception of the mediastinum, although they may arise within the lung. It is not rare for them to encroach on one or both pleural cavities but for a dermoid cyst to produce symptoms which seem to be in a pleural cavity without any obvious evidence of a mediastinal counterpart is rare. Harrington, in 1937, reported sixteen cases of mediastinal dermoid tumors from The Mayo Clinic. In none of these was the clinical course similar to that in the case which we are reporting.

The patient, a youth fifteen years of age, was first seen at The Mayo Clinic May 29, 1939. His trouble began in 1925 when he was four years old with an acute illness which was diagnosed pneumonia. After this postpneumonic empyema was thought to have developed on the right side and drainage of the right pleural cavity was instituted. Drainage gradually subsided and after a period of about two months the wound was entirely healed and the patient seemingly was restored to normal health.

The patient was free of symptoms for nine years. Then after a period of anorexia, sweats, chills and fever, a diagnosis of chronic empyema of the right side was made elsewhere and right thoracotomy was performed for drainage. The patient's father said that at that time considerable yellow cheesy material drained from the wound. In the next five years four more operative procedures were performed to facilitate drainage.

General examination at the clinic revealed nothing abnormal other than the condition in the thorax and slight emaciation. Early clubbing of the nails was noted. There was a draining sinus on the right in the posterior lateral portion of the chest wall which contained a rubber tube. The patient was 64 inches (162.6 cm.) in height and weighed 95 pounds (43 kg.).

*From the Division of Surgery, The Mayo Clinic (Gray), and the Mayo Foundation (Woodruff), Rochester, Minnesota.

The value for hemoglobin was 11.4 gm. per 100 c.c. of blood. Erythrocytes numbered 3,850,000 and leukocytes 12,000 per cubic millimeter of blood. A roentgenogram of the thorax revealed the drainage tube in place and thickened pleura on the right (Fig. 1).

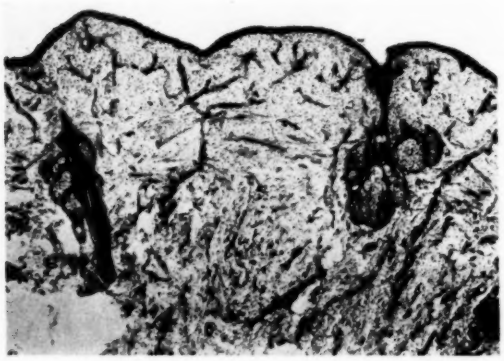

Fig. 3. Squamous epithelium and the skin appendages, hair follicle and sebaceous glands, which are diagnostic of a dermoid, are well shown (× 50).

A diagnosis of chronic empyema of the right lung was made and on May 30, 1939, surgical intervention was instituted. The sinus was explored with a probe and was found to extend outward and upward for approximately 18 cm. Accordingly, a portion of rib immediately over this sinus was removed and the cavity unroofed. A large quantity of purulent material mixed with a caseous looking substance was scooped out. A cavity which measured about 10 cm. in diameter was revealed. Several peculiar looking rounded tumors, covered with skin and hair, were firmly attached to the inner and lower portions of the cavity (Fig. 2). Communicating with the main cavity in the mid-portion was a small cavity which probably represented an empyema pocket associated with the dermoid cyst.

As much of the contents as possible were removed from the cavity but it was impossible to remove all of the wall of the cyst. Consequently, the lining was destroyed with phenol and then the part treated was well cleansed with alcohol. The communication between the cyst proper and the empyema pocket was enlarged in order to establish free drainage. The whole cavity was packed lightly with plain gauze. The pathologist reported the tissue removed to be a dermoid cyst lined by squamous epithelium with hair follicles and sweat glands and containing sebaceous material. A section from one of the pedunculated growths demonstrated clearly the fact that this intra-thoracic lesion contained all of the elements of the skin (Fig. 3).

The patient's postoperative course was uneventful. A transfusion of 500 c.c. of citrated blood was administered on the second postoperative day as a supportive measure. The drainage gradually subsided and the cavity slowly began to fill in with healthy granulation tissue. The patient was dismissed from the clinic July 5, 1939, and at that time the cavity had filled in partially with clean granulation tissue and there was no sign of any remaining portion of dermoid tissue.

Since dismissal reports have been received from the patient and his attending physician. Convalescence was satisfactory and the boy rapidly gained weight and strength and soon was able to engage in all the activities of his associates. The cavity continued to fill in and when the last report was received in February, 1940, there was only slight drainage.

The ultimate prognosis in this case must be guarded because it is possible that a remnant of the cyst wall has escaped destruction by the phenol. However, the chronically infected cyst which was gradually undermining the patient's health has been eliminated and the immediate prognosis at least should be favorable.

Although an infected tumor simulating chronic empyema is undoubtedly rare, the possibility of such a syndrome should be considered in cases of a persistent draining from the thorax. Not infrequently chronic empyema may be allowed to persist indefinitely because adequate drainage of the empyema cavity has not been effected. The exact nature of the lesion in this instance was determined only after adequate drainage had been established.

OBSERVATIONS ON THE OCCURRENCE AND PREVENTION OF SUDDEN DEATH

JOSEPH F. BORG, M.D.

Saint Paul, Minnesota

THE frequency of unexpected sudden death calls attention to this condition as a problem to be studied from the aspects of occurrence, mechanism and prevention. It may occur in individuals apparently in good health or in patients whose conditions give no indication that such an outcome might occur. Since a structural defect is only exceptionally found to account for it, it has been logical to consider it as predominantly a cessation of cardiac function on a physiologic basis.

Under two known mechanisms, ventricular standstill and ventricular fibrillation, the heart may suddenly cease functioning. These may be transient but ventricular fibrillation is rarely so. Experimentally, Hering[3] was unable to produce death by ventricular standstill. In patients with Stokes-Adams syndrome, the syncopal attacks are nearly always due to transient ventricular standstill, and it is probable that sudden death in these patients may be explained on that basis. This group accounts for a very small proportion of those dying suddenly.

Ventricular fibrillation, however, rarely reverts to an effective ventricular rhythm and it has therefore been considered by many to be the most likely cause of sudden death. Animal experiments and clinical observations in recent years have tended to verify this opinion and much has been done to explain the mechanism by which this accident occurs. Thus Hering[3] claimed that ventricular fibrillation in animals was an irreversible process, with lethal outcome. It is well known that experimental ligation of a coronary artery will produce ventricular fibrillation. Furthermore, the occurrence of frequent ventricular premature contractions and ventricular tachycardia shortly before sudden death in coronary thrombosis suggests that the latter accident is due to ventricular fibrillation, especially since these irregularities have been described by Schwartz[13] and his coworkers as prefibrillatory arrhythmias announcing the imminence of transient ventricular fibrillation in the cases they reported.

Of great importance have been the recent contributions which indicate that the intimate mechanism of sudden death is to be explained on the basis of sympathetic nerve stimulation of a myocardium sensitized by some toxin or disease state. In view of the evidence that general sympathetic nerve stimulation acts through chemical mediation of an epinephrin-like substance produced at its site of action, it seems probable that the innervation of the myocardium acts likewise. This view is supported by a number of observations by various investigators. Thus, Rothberger and Winterberg[12] found that in dogs partially poisoned with barium chloride, sympathetic nerve stimulation or the use of epinephrin would produce ventricular fibrillation. Otto[11] showed that the ventricular fibrillation caused by coronary ligation in dogs was prevented by section of the sympathetic fibres. Hooker[4] has reported experiments in dogs in which areas of fibrillating ventricular myocardium were cut away from the heart with a resulting cessation of the fibrillation, even a return to rhythmic contraction being recorded. While he did not attempt to draw the inference from these experiments, it seems likely that some substance was being elaborated by nerve action on the myocardium and resulted in ventricular fibrillation which stopped in a segment of the heart muscle when it was cut away from the whole. Whether this substance is identical to epinephrin or is an epinephrin-like substance (sympatheticomimetic amine) it is probable that it plays an important rôle in the production of ventricular fibrillation in human subjects.

This conception of the mechanism of ventricular fibrillation leads to the consideration that sudden death due to this cause might be preventable if it were possible to select those patients who after clinical study were considered liable to this condition and if some measure were available to counteract the action of epinephrin or reduce the irritability of the heart muscle. An attempt to throw some light on these two problems is made in the present study.

Concerning the first, a study was made of the records of all patients who, among the 22,490 admissions to Ancker Hospital during 1934-1935, died suddenly, unexpectedly and with no indicated or proved structural defect to account for

such outcome. (Autopsies were performed on 50 per cent of these patients). From this analysis certain facts emerged which indicate the type of patient in which this accident has a tendency to occur. These will be discussed.

Concerning the second, recognition was given the probable rôle of epinephrin or an epinephrin-like substance in the mechanism of ventricular fibrillation. Nathanson has shown that no satisfactory antagonist to epinephrin can be used clinically. However, it is possible to depress myocardial irritability, and in view of the effectiveness of quinidin in the treatment of auricular fibrillation, it has been used therapeutically and experimentally with apparent success in producing this effect in conditions of known myocardial irritability. It has been considered successful in preventing ventricular arrhythmias (Scott, Levine and Fulton).[14] Cases have been reported (Dock,[1] Escamilla[2]) where quinidin seemed to prevent attacks of paroxysmal ventricular tachycardia. Levine[6] and Jackson, Friedlander and Lawrence[5] showed that quinidin inhibited experimental ventricular fibrillation in animals. Nathanson[9] in experiments on human subjects, was able to inhibit by quinidin the onset of pre-fibrillatory rhythms which were produced by sympathomimetic amines. Morawitz and Hochrein[8] used quinidin empirically with success in an attempt to prevent sudden death. These contributions indicate that quinidin modifies the action of the substance which presumably produces ventricular fibrillation, and provides a rational basis for the possibility of attempting to prevent such a development in individuals who may be considered liable to sudden death.

The present study is based on an analysis of the cases of sudden unexplained death in a large general hospital (Ancker Hospital, Saint Paul) over a three-year period, 1934-1935-1936, during the third year of which quinidin sulphate was administered to patients who seemed liable to sudden death as shown by the experience of the first two years. The series consists of fifty-one patients in whom death was unexpected at the time, sudden in its appearance, and unexplained on a basis of structural change. Omitted therefore were the sudden deaths due to rupture of the heart, ruptured aortic aneurysm, and other conditions where demonstrable cause for this type of death was present. Autopsy verification of the diagnosis was obtained in twenty-five of the fifty-one cases. As shown in Table I, this material is derived from a total of 2,570 deaths occurring in a total of 33,243 admissions to Ancker Hospital, Saint Paul, during this period.

TABLE I. UNEXPLAINED SUDDEN DEATHS—ANCKER HOSPITAL

Diagnosis	1934	1935	1936
Coronary Sclerosis	8	3	0
Cor. Scler. + Hypertensive Heart Disease	3	6	3
Coronary Sclerosis + Aortic Stenosis	3	4	0
Coronary Thrombosis	5	4	1
Hypertensive Heart Disease	1	3	0
Aortic Stenosis	0	1	0
Syphilitic Aortitis	1	1	0
Miscellaneous Group	2	1	1
Total Admissions	11,036	11,454	10,753
Total Deaths	896	786	888
Sudden deaths	23	23	5
(Per Cent of Total)	(2.57%)	(2.93%)	(.56%)

While the number of cases is not large, certain facts stand out in a study of the 1934 and 1935 sudden unexplained deaths (twenty-three such fatalities each year) which were helpful in indicating the patients most liable to such exitus.

Sex.—Divided according to sex, these cases show a great preponderance of males. In 1934, there were twenty males and three females, and in 1935, nineteen males and four females.

Age.—A marked tendency for these deaths to occur at advanced ages was demonstrated, 76.1 per cent occurring after the age of sixty, with the peak decade being that of the seventies.

Electrocardiography.—Electrocardiograms were recorded in twenty-one of the cases. All of these showed abnormal T waves and eight showed QRS abnormalities, notching, slurring and widening.

Congestive Failure.—As might be expected in a group of hospital patients congestive failure was not uncommon, although all cases in which this condition was the direct cause of death were eliminated. Twenty-nine were in congestive failure when sudden death occurred and seventeen patients were in a state of good compensation.

Arrhythmias.—Since it is known that arrhythmitas, especially premature contractions, precede the onset of ventricular fibrillation, it is of interest to note that fourteen (30.4 per cent) of

the patients showed numerous ventricular premature contractions. Auricular fibrillation was present in seven (15.2 per cent) of the patients.

Diagnosis.—In all except two cases a definite diagnosis of heart disease was made (Table I). The exceptions might possibly have been included had autopsies been permitted. These were patients with involutional melancholia, aged fifty-seven, and convalescent hemiplegia, aged seventy-two. Degenerative heart disease is seen to be outstanding, coronary sclerosis alone or associated with hypertensive heart disease, coronary thrombosis, or aortic stenosis accounting for 78.3 per cent of the patients. This does not present the real incidence of coronary sclerosis since one patient in a miscellaneous group, an asthmatic, showed coronary disease at autopsy, and other deaths occurred at ages where such condition is common. Another observation of interest relates to the diagnosis of aortic stenosis. This was made in eight instances, in seven of which there was a complicating coronary sclerosis. All of the seven patients were between fifty-eight and eighty-eight years of age. Sudden death in uncomplicated hypertensive heart disease ranked relatively low in the group, only four cases being found. Syphilitic aortitis contributed two cases, and the miscellaneous group is composed of one case each of asthma, hemiplegia, and involutional melancholia.

In view of the foregoing experience of the years 1934 and 1935, with the coöperation of the various hospital services, it was decided to administer during 1936 quinidin sulphate three grains three times daily to all patients with degenerative heart disease, aortic stenosis or syphilitic aortitis upon admission to the hospital. The drug was given in addition to any other indicated therapy and stopped if not well tolerated.

The figures presented for this year show a marked decrease in sudden deaths although the general hospital mortality was higher than in the previous years. Only five deaths occurred, four in patients with coronary sclerosis complicated by hypertensive heart disease or coronary thrombosis with congestive heart failure, and one in a patient with advanced pulmonary tuberculosis on whom an autopsy was refused but no recognized cardiovascular disease had been found. Of the four cases with degenerative heart disease quinidin had not been given in three. A brief review of these five histories is presented as follows:

Case 1.—A woman, aged sixty-five, was admitted with severe dyspnea, substernal pain, auricular fibrillation and shock. Onset had been sudden and diagnosis on a previous admission was hypertensive heart disease, electrocardiogram showing left axis deviation and inverted T waves in lead 1. Admission diagnosis was coronary thrombosis. The patient died suddenly twelve hours after admission. Quinidin was not administered.

Case 2.—A man, aged seventy-two, was admitted complaining only of pain in the left foot. The diagnosis of coronary sclerosis and diabetes complicated by gangrene of the left foot was made. The electrocardiogram showed auricular fibrillation, QRS notching and T wave inversion. Compensation was good. The patient died suddenly in bed. Quinidin was not administered.

Case 3.—A woman, aged seventy-two, was admitted with edema, dyspnea, cyanosis and auricular fibrillation. A diagnosis of congestive heart failure on basis of hypertensive heart disease and coronary sclerosis was made. Quinidin was administered. Clinical course was not favorable, but the sudden termination was unexpected. Diagnosis was verified by autopsy.

Case 4.—A woman, aged forty-five, complained of nocturnal dyspnea and precordial pain on exertion. Clinical diagnosis was hypertensive heart disease with auricular fibrillation. No congestive heart failure was present. The electro-cardiogram showed QRS and T wave abnormalities. Quinidin was not administered. The autopsy showed cardiac hypertrophy, coronary sclerosis, myocardial fibrosis and cerebral malacia.

Case 5.—Male, aged fifty-four, was a patient on the tuberculosis pavilion with no significant complaints and heart disease was not suspected. Clinical diagnosis of advanced pulmonary tuberculosis was made. Autopsy was not performed.

From these results it is obvious that the desired administration of quinidin to all patients for whom it was intended was not entirely accomplished. While the total number of omissions was not determined there is reason to believe that it was small. It is of particular interest therefore to note that only one of the patients who died suddenly in 1936 had received the drug, and the question may be properly raised as to whether or not the rather advanced state of congestive heart failure here should not have contraindicated its use.

Discussion

Inasmuch as it is impossible to state what percentage of patients suffering from heart disease died directly from this cause it is therefore im-

possible to determine what proportion of these die suddenly. However, some idea of the importance of the subject can be obtained from the studies of Nathanson[10] who found that 60 per cent of 113 patients dying of occlusive coronary disease died suddenly. These figures are high because they include deaths outside of the hospital, much more often sudden than in the hospitalized group in this class of patients. Nevertheless they emphasize the problem as more important than its casual consideration would suggest.

While it appears that the majority of the sudden deaths in the years surveyed were not associated with congestive heart failure, these figures may be misleading. The state of compensation described was that present at the time of death, while many of the compensated patients had been decompensated on admission to the hospital. The present study at least suggests that sudden death occurs more often in those without congestive failure.

The general hesitancy in the use of quinidin in congestive heart failure merits consideration as to whether its use was justified in such patients included in this study. Unless Case 3 could possibly be considered as a quinidin death, no patient who had received the drug in 1936 died suddenly or developed the arterial emboli so feared by some investigators.

The desirability of the use of quinidin in an attempt to prevent sudden death is open to discussion. Considerable difference of opinion exists as to the justification for the use of a drug continuously over a patient's lifetime when a certain clinical picture presents itself. Much can be said on both sides. As far as risk is concerned in the continued prophylactic use of quinidin no known well controlled studies point to danger, and the present study would not indicate such to exist. The psychologic effect on the patient of the use of a drug designed to prevent sudden death would be most unfortunate if the purpose were made known, although the matter of prolonged administration should not be a serious objection. On the other hand this study would suggest that such a prolonged regime of quinidin administration may in many instances extend a comfortable and useful existence for many patients with heart disease. Further studies should be made of this subject before any crystallization of opinion can take place.

Conclusions

Results obtained by the use of quinidine sulphate in the present study furnish clinical support to the belief that ventricular fibrillation is the usual cause of sudden death.

Observations are made which support the suggestion that quinidine sulphate may be effectively used in preventing sudden death in patients liable to it.

This study indicates that the patients in whom sudden death is most apt to occur are of advanced age, suffering from degenerative or syphilitic heart disease, showing abnormality of the electrocardiogram and frequently manifesting arrhythmias.

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WHAT'S WRONG WITH THE PATIENT WHO IS ALWAYS TIRED?*

WALTER C. ALVAREZ, M.D.
Rochester, Minnesota

EVERY week I see a number of patients whose main complaint is that they are tired and exhausted. They have no "pep," and the least exertion makes them feel worn out. Many complain that they cannot do their work properly and they can't get much fun out of life. The stenographer or clerk can't go out with her beau in the evening and the wife can't go anywhere with her husband and enjoy it. The school teacher can barely get by until Friday, and with her it is always a race between a nervous breakdown and the end of school in December and May. We physicians are all seeing these persons, and we all wish often that we knew what is wrong with them.

From the letters from prominent physicians which many of these patients bring with them, I gather that the medical profession today is inclined to look on these complaints as due to some localized disease or focus of infection somewhere in the body. As a result the patients are put through one expensive and exhaustive laboratory and roentgenologic examination after another. All possible foci of infection are removed or treated, and occasionally in desperation a surgeon will explore the abdomen and remove the appendix.

When nothing is found, the patient moves on, and the next clinician promptly puts the patient through another complete examination. He and the patient both hope that some local disease will be found which was missed before. Unfortunately the pressure on the physician to find some local cause is so great that he is likely to grasp at whatever diagnostic straw is offered and to make all he can of one basal metabolic rate of —15, one blood pressure reading of 105 mm., one blood sugar determination of 85 mg. or a little spasticity of the colon.

All this does not mean that when a patient who has been strong and well begins to feel tired out a careful examination should not be made. It most certainly should be, and this is particularly true when the failure in strength and energy and the loss of a sense of well-being come to a man or woman past middle age who has previously enjoyed good health. In such cases it usually means the coming of some serious disease, and the physician must hunt through the body for carcinoma or for signs of pernicious anemia, hypothyroidism, hypertension, diabetes, or a failing heart or kidney.

If the fatigue and loss of "pep" and interest in life come suddenly in a person past middle age, the cause is almost certainly a small stroke. Curiously, physicians rarely think of this possibility when the thrombosis does not happen to involve the centers for speech or for arm or leg. Commonly the episode is thought to be due to an "acute indigestion" because it is so often associated with dizziness and a storm running down the vagus nerves to produce vomiting and abdominal discomfort. Sometimes careful history taking will show that there were several of these small episodes. Often they come in the morning when the patient wakes. Especially after several of them there is likely to be some loss of memory, a loss of interest and zest in life, and perhaps an inability to work. Unfortunately, this sudden and marked change in the temperament of the patient is seldom mentioned when the victim and his family are in the physician's office. The story must be dug out. It is important that the physician recognize the true nature of these little upsets because then he will know that it is useless to try to help the patient or to cheer him up. His brain is injured and he cannot be helped.

In younger patients the physician will, of course, check the lungs carefully. Occasionally the main symptom of chronic appendicitis in a previously healthy college student will be a mental slowing up and a feeling of toxicity and loss of "pep." If the physician can then dig out a story of one or more attacks of cramping abdominal pain, appendectomy is indicated. Occasionally a tired youngster will be found to have hypertension or hyperthyroidism. Young women are often worn down by the recurrence of episodes of painful menstruation. They do not recover completely from one spell before they are into another. Other young women are anemic from repeated flooding. The blood must always

*From the Division of Medicine, The Mayo Clinic, Rochester, Minnesota. Read before the meeting of the Minnesota State Medical Association, Rochester, Minnesota, April 22 to 24, 1940.

be examined carefully to rule out a beginning leukemia. Especially if a little fever is noted on some days it will be wise to see if the patient's serum agglutinates Brucella abortus in high dilutions.

Often the story will be that the fatigue state followed an influenza, and then in rare cases the physician may think of the possibility that there was a mild encephalitis. More commonly, after a respiratory infection, there will be a mild generalized arthritis or fibrositis; the patient will ache all over, and with this there will be a feeling of fatigue and toxicity. Occasionally the story will be that the patient thought he was poisoned by tainted food, and after the diarrhea stopped he was left with an irritable, gassy bowel and a tendency to diarrhea. In these cases one will suspect that a small intestinal enteritis has not entirely cleared up.

In most cases in which the patient is young or middle-aged, none of these things are found, and then the question arises: Did the patient overwork badly or did he have enough strain, unhappiness, sorrow or insomnia to break down the nervous system of a normal person? One sees this type of breakdown often in the case of a woman who has had several children in rapid succession. Perhaps she has been without a maid, and perhaps a mother-in-law in the home has been adding to her strain. Perhaps she had an operation, an auto accident, a difficult labor, or a bad attack of influenza, and then tried to go back to work too soon. Or the patient may be a shop girl whose hours are too long, or she is a factory girl whose work is too heavy, or the victim may be a hard-driving business man or a too busy physician who has never had a vacation. In all these cases, instead of repeating the examinations indefinitely, it seems to me more logical to make the patient take a rest and then see if there are any symptoms left.

In another large group of cases one will find that the patient was frail and asthenic to begin with, and then had heavier burdens put on him or her than could be borne. In such cases it will be obvious why the patient got into trouble.

Finally, there is a large group of tired patients who, so far as one can find, have not been under any unusual strain. They seem to break down only because the tendency was lying latent in the nervous system, inherited from some ancestor. Often bad psychic habits bring on the crash. Thus, I remember once asking a fine looking young woman with a lovely home and a nice husband why she was on the edge of a nervous breakdown. I wondered what was wearing her out. Her answer was, "I wear myself out." As I came to know her I found how true this was. She was the type of psychopathic person who can't make a decision even on minor matters, or if at last she makes one she can't stick to it. Her sister was insane, and her tendency to indecision was apparently her share of the family curse.

As I said, many of the persons in this group wear themselves out by using their brains unwisely, uneconomically, and to no useful purpose. In one hour of frantic worrying, or in one riot of emotion over some little happening, or in one "post mortem" over some unpleasant experience or ancient family row, a woman will use up more energy than a calm sensible person uses in a day. Some of these persons are so psychopathic, touchy, shy, diffident, or irritable that all the problems of working and living and adjusting to contact with their fellows are difficult and wearying. Others, and particularly women about the age of forty, are perfectionists: They want everything just so, and they wear themselves out trying to reform husband and to make the home spotlessly clean.

But every so often the physician will find a tired and depressed patient who apparently hasn't many bad mental habits, who hasn't had obvious strain, and who has had no grief or cause for worry. What then? Then if the physicion will only take the trouble to get a good history, often from a relative, he is likely to find that one or more of the patient's close relations have suffered with melancholia or depressions of some kind. In many such cases there can be no question that the patient's real difficulty is a mild depression. It represents what he or she got out of the family grab-bag of inheritance. One can be even surer of this when one can get the patient to tell how between periods of depression he or she has been too energetic and talkative and ambitious. Such persons have a cyclic temperament with their ups too high and their downs too low. Fortunately, in most of these cases the defect that sent a mother or an aunt to the insane asylum was diluted by good genes from the other side of the family, and hence such depressions are compatible with the maintenance of a useful life.

It always saddens me to see these persons who have so definitely a psychiatric problem being put through all sorts of medical overhaulings and being treated strenuously for amebiasis, low blood pressure, low blood sugar, or low blood calcium, or perhaps being operated on in the hope that some causative lesion will be found in the abdomen. It is unfortunate that we physicians at college had practically no training in psychiatry, and hence we are so unprepared to recognize the insanity of those many persons who remain quiet and well behaved. Worse yet, when consulted by these patients, clinicians will often ignore or ridicule the diagnosis of insanity after it has been made for them. I have seen them keep ignoring it until the patient finally gave up and took refuge in a sanatorium for "mental cases."

The point I would like to emphasize is that when a patient is depressed and apparently exhausted without obvious cause, when two or three overhaulings have failed to reveal any organic disease, and when one or more near-relatives have suffered with melancholia, the physician should begin to suspect that the trouble is a mild depression.

Summary

Many patients complain primarily of fatigue and easy tiring. They get put through one thorough examination after another with the hope that some local disease or focus of infection will be found. Usually this isn't found. Naturally one careful examination should be made, especially when ill health comes after middle age to a person who has been previously well. Then one may find serious disease such as cancer, anemia, hypertension, diabetes, hypothyroidism, or a failing heart. If in older persons depression, fatigue, loss of interests, change in personality, and an inability to work come suddenly, the cause is probably a slight stroke. In the case of college students, ill health with indigestion and feelings of fatigue may be due to subacute appendicitis.

In many cases nothing is found on thorough examination. Then the physician must be careful not to grasp at diagnostic straws. Then he must see if the patient has had enough strain, unhappiness, sorrow or insomnia to account for the situation. In many cases, with or without strain, a person with a psychopathic inheritance breaks down. In some cases the patient's fatigue and nervousness represent an equivalent of melancholia in a near relative. It is most unfortunate that mild melancholia is today rarely recognized by clinicians. The average physician today has little ability to recognize the patient with borderline insanity.

POLYNEURITIS WITH FACIAL DIPLEGIA (NEURONITIS) FOLLOWING SERUM SICKNESS IN AN ADULT

GORDON R. KAMMAN, M.D., F.A.C.P. and M. WEISBERG, M.D.

Saint Paul, Minnesota

DESCRIPTIONS of the syndrome of polyneuritis with facial diplegia have appeared in medical literature under a variety of names. Different authors have referred to the disease as acute febrile polyneuritis, curable polyradiculoneuritis with albumino-cytologic dissociation, radiculoneuritis, meningoradiculoneuritis, myeloradiculitis, et cetera. Probably the first description of the disease in English was given in 1898 by Mills[2] who called it "neuronitis." Since then various authors have used this term but among neurologists controversy exists as to the proper name for the syndrome. We prefer the clinical designation of polyneuritis with facial diplegia for the reason that the pathological changes in the central nervous system are variable, and any nosological designation embodying a description of these changes would not be sufficiently broad to include all cases.

In spite of the confusion in the literature over what this condition should be called, the clinical picture is a fairly constant one. The disease in the majority of cases is preceded by a mild transitory infection which may represent the stage of invasion of the causative agent. There then is a latent period of from one to fourteen days, followed by the appearance of signs of polyneuritis predominantly of the motor type. Some cases show mild sensory changes in the early stages, some have fibrillary twitchings, and in some there

is impairment of vesical or rectal function or both. The weakness and parasthesias usually begin in the lower extremities, involving the muscles of the trunk and upper extremities to a lesser degree in from one to four days. In about one-third of the cases facial paralysis of the nuclear type is present[1] and we have seen choked discs in a few cases.

The most constant feature of polyneuritis with facial diplegia is the albumino-cytologic dissociation in the cerebrospinal fluid. Although we have seen cases in which there were as many as 30 lymphocytes per cubic mm. of cerebrospinal fluid, the cell count in the great majority of cases is within normal limits. There *always* is an increase in the amount of spinal fluid protein. This increase might be slight, ranging around 50 mg. per 100 c.c., or it may be enormous reaching 1200-1500 mg. per 100 c.c. Without this albumino-cytologic dissociation one is not justified in making a diagnosis of polyneuritis with facial diplegia or of neuronitis.

The course of the disease is usually benign and recovery occurs in from six months to two years. Death may occur from respiratory paralysis, intervening sepsis, or general debilitation. In a series of thirty-five cases, Gilpin et al[1] encountered a mortality of 14 per cent. This is somewhat higher than our experience.

The number of pathological studies has been quite limited but Gilpin et al[1] found the pathological changes to be limited to the peripheral nerves and the dorsal root ganglia. The anterior horn cells as well as the rest of the spinal cord is not involved in the process which consists mainly of a patchy degeneration of the myelin with fragmentation of the axis cylinders of the peripheral nerves, without any evidence of inflammation.

The case we are reporting is unusual because the onset was not preceded by the usual type of infection, and the course of the entire illness was only a few weeks.

S. K., an unmarried male, thirty-two years of age, was a grocery clerk. His family history was irrelevant. He had acute rheumatic fever in 1913, a tonsillectomy in 1917, and influenza in 1919. No history of allergy in either the patient or his family could be elicited. Mr. K. was well until June 17, 1939, when at about 3 P. M. he stepped on a nail and sustained a puncture wound of the sole of the right foot. He applied tincture of iodine to the wound and continued to work. A few hours later, he began to have pain in the foot. Several hours later, one of us (M. W.) was consulted and magnesium sulphate foot baths were prescribed. About thirty-six hours following injury, the patient received 1,500 units of tetanus antitoxin subcutaneously into the left upper arm. The foot improved and the patient returned to work on June 20th. He felt well the next day, June 21, but noticed a few hives over both upper extremities. That evening he had a chill and the next morning complained of aching in all his joints and he felt feverish. He went to work but about 4 P. M. had to stop because of general malaise. The temperature rose to 103 degrees. On June 23 the hives became more generalized and he stayed in bed. Two days later, June 25, he had diarrhea with vomiting, and temperature of 102.6. He remained in bed for four days and gradually improved. On June 29, his feet became numb and the next day both hands were numb but there was no muscular weakness. The numbness ascended to the wrists and knees and on July 1, fifteen days following injury, patient complained of backache. On the morning of July 4, he awoke with pain in back of neck, jaw and face. That afternoon he developed a paralysis of the left side of his face, and was admitted to Bethesda Hospital in Saint Paul.

Upon admission Mr. K. had a left facial paralysis of the peripheral type. The cranial nerves were otherwise normal. There was weakness of both grips and the supinator reflexes were absent. The muscles of the lower extremities showed a flaccid weakness of all groups. Deep reflexes were absent but no pathological reflexes were elicited. Touch, pain, passive motion and vibration sense were diminished in the hands and feet. The laboratory findings were as follows: Hemoglobin 78 per cent, red blood cells 4,070,000, white blood cells 8,400. Differential count normal. Urine normal. C.S.F. was under normal pressure, contained 155 mg. protein per 100 c.c., 7 lymphocytes per cubic mg. The Wassermann reaction was negative and the colloidal gold curve was flat. Temperature remained normal throughout patient's stay in the hospital.

During the first four days in the hospital Mr. K. became worse. The right side of his face became paralyzed and he had a complete facial diplegia. The right triceps jerk disappeared and the muscles around the shoulder girdles became weak. Pilocarpine sweats, potassium iodid by mouth, and daily subcutaneous injections of thiamin chloride were prescribed and in about three weeks definite improvement could be noticed. Sensation and muscle strength improved, the deep reflexes returned to normal and on August 9, 1939, the patient was released from the hospital with only a slight weakness of the left side of the face. On August 30, the examination was negative except for a slight weakness of the left side of the face and Mr. K. was permitted to return to full time work.

Comment

While the association between the serum sickness and the polyneuritis may be wholly fortuitous, it is not unreasonable to suppose that the serum sickness was the precipitating factor in

the disease. The possible rôle played by allergy in this condition has never been determined, but there are those who think that many of the more obscure types of involvement of nervous parenchyme are allergic in nature. In our case we were dealing with a perfectly healthy, robust, young adult male. He had not had a recent infection of any kind. Directly upon the subsidence of an attack of serum sickness which followed the prophylactic administration of tetanus antitoxin, he developed the early symptoms and, subsequently, all of the typical symptoms of polyneuritis with facial diplegia. The only way in which the course of the disease differed from the classical picture was that it was very much shorter.

This patient was injured during the course of his employment and the question of compensation was raised. Because of the close temporal relationship between the serum sickness and the involvement of the nervous system, and because of the absence of any other demonstrable etiological factor in the disease, compensation was granted. One of our main purposes in reporting this case is to stimulate others to report cases of a similar nature should they occur.

Summary

1. Polyneuritis with facial diplegia (neuronitis) is a well established clinical entity, the etiology of which is obscure.

2. In the majority of cases the symptoms are preceded by a mild transitory infection.

3. A case is presented in which the symptoms appeared following an attack of serum sickness due to the prophylactic administration of tetanus antitoxin.

4. The patient was injured during the course of his employment and was granted workmen's compensation.

1044 Lowry Medical Arts Building.

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PRIMARY ADENOCARCINOMA OF THE APPENDIX AND CARCINOID TUMORS

F. C. SCHULDT, M.D., F.A.C.S.
Saint Paul, Minnesota

R. S., aged sixty-four, was seen July 18, 1938, complaining of a sore spot on the right side of the abdomen just above the iliac crest. He had had pain of moderate severity for the preceding three days without nausea. For the last two days he had had no appetite. The pain in the abdomen was increased on deep breathing and coughing.

On May 15, 1937, I had seen him in the office when he consulted me concerning a pain in the right abdomen of two week's duration. At one time the pain was completely relieved after a laxative. On examination at this time there was no tenderness at all over the appendix area, but there was a tenderness above the right iliac crest. White blood cells were 11,000. Three days later the pain was gone and on examination there was no tenderness. I did not see the patient again for fourteen months. There had been no weight loss. No definite diagnosis was made at this time.

On July 18, 1938, his temperature was 99°; urine, normal; heart, normal. There was slight tenderness over McBurney's point and marked tenderness above the crest of the right ilium. White blood cells were 14,250.

A diagnosis of an acute retrocecal appendicitis was made.

On July 19, 1938, under special anesthesia, a right rectus incision was made. As soon as the abdomen was opened free thin pus was found. The appendix was retrocecal. The head of the cecum and all of the intestines showed a general peritonitis. The appendix was removed in two pieces. The appendix lay wide open in its mid-portion and at this point the tissue of the appendix had a peculiar tumor-like appearance (Fig. 1). The appendix was gangrenous at its distal portion. The abdomen was drained.

Pathologic report: appendix was 10 cm. long; pus exudate in proximal portion. Near the mid-portion was a gelatinous tumor mass. Section showed a suppurative appendix and also an infiltrating epithelial tumor, portions of which had undergone gelatinous change with some differentiation into glandular elements (Fig. 2).

Diagnosis: 1. Adenocarcinoma with gelatinous degeneration. 2. Perforation with suppurative peritonitis.

Suction decompression was necessary, postoperatively. Convalescence was then normal and the patient was quite well.

On April 3, 1939, he complained of general pain in the abdomen after eating a heavy meal. He felt bloated but obtained relief after a satisfactory stool. A hard nodule was found at the upper end of the incision.

By May 8, 1939, he had lost some weight. The nodule in the fascia of the incision was larger and more tender. It was recognized as a possible metastasis and he was advised to have it removed.

At the time of operation the malignancy was recog-

nized while the appendix was *in situ* and the utmost care was used to prevent implantation. No forceps were used to clamp the appendix. But our exhibition consists of a ruptured appendix, a rupture due to a neoplasm. As the abdomen contained considerable free pus the possibilities of carcinoma cells being widespread had to be considered.

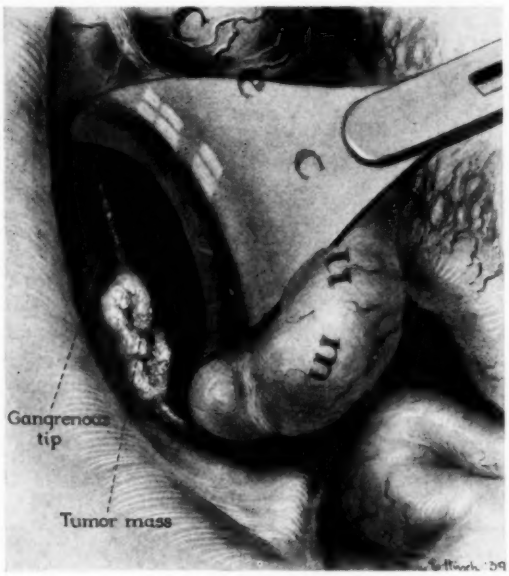

Fig. 1. Drawing showing appearance of appendix at operation.

On June 1, 1939, this nodule, which measured 3.5 cm. in diameter, was excised under local anesthesia. The nodule was not infiltrating and could be lifted while it was being removed from the rectus muscle. On gross section it appeared malignant and on microscopic section proved it to be a gelatinous adenocarcinoma.

Following this ascites required the tapping of three to four litres of a straw-colored fluid every ten to twenty days. The fluid became eventually definitely bloody. There was a steady loss of weight and constant pain throughout the abdomen. No masses could be felt after the fluid was removed, but another nodule formed in the incision.

Discussion

The pre-operative diagnosis in this case of carcinoma of the appendix was acute retrocecal appendicitis. All references in the literature stress the point that there has been no pre-operative diagnosis made of carcinoma of the appendix.[3]

In our case the carcinomatous involvement had caused a perforation and thereby an acute peritonitis. The malignancy was recognized before the appendix was removed from the abdomen. All of the small intestine and the cecum had a surface injection like that of an acute peritonitis. As the cecum was retracted medianwards the appendix became visible with its proximal third injected, its middle third nodular and neoplastic and the terminal portion discolored and gangrenous.

Waugh and Findley[11] report a case of mucocele of the appendix which had degenerated into a carcinoma and then ruptured with a consequent generalized metastatic process of the whole pelvis. Our case is a parallel to this as here a carcinoma had been growing for some time until it ruptured through the malignant area without producing the classical picture of a ruptured appendix.

In the classification of the neoplastic processes occurring in the appendix the following four forms are to be recognized:

1. Appendicitis fibroplastica, the huge thick walled appendix.
2. Mucocele of the appendix, the occasional result of lumen obliteration, and rarely degenerating into malignancy.
3. Carcinoid tumor of the appendix, called by Aschoff[10] "mucous membrane naevus," rarely showing metastases.
4. Gelatinous adenocarcinoma primary of the appendix; rapidly metastasizing.

The adenocarcinoma is of the gelatinous type and is the most common form found in all malignancies of the colon. The structure shows a scarcity of cells but an abundance of a gelatinous mucus.

It would be interesting to know just what pathologic picture was present fourteen months previous when the patient complained of indefinite pain in this area. At that time his symptoms disappeared upon correcting his constipation. Is it probable that he also had a mucocele following a lumen obliteration which degenerated into a malignancy? We can not prove or disprove this, but on careful examination of the removed specimen there were only solid tumor masses having a glassy appearance (the gelatinous structure).

It is probable that neither a mucocele nor a primary carcinoma nodule causes any pronounced symptoms and consequently no medical advice is sought. A rupture immediately precipitates an acute abdomen. In the carcinoid tumors[8] the symptoms are those of an interstitial appendicitis of the chronic type.

The first authentic case of primary carcinoma

of the appendix is reported by A. Beger[1] in 1882. In a splendid article he recites the details of his case.

In a forty-seven-year-old man a "boil" appeared on the skin surface in the right inguinal region. This was opened and drained of an odorless pus with no gas or fecal material. Three and one-half years later the patient was operated upon under chloroform anesthesia with salicylic spray used for asepsis. This proved to be a carcinoma of the tip of the appendix, which had grown into the abdominal wall and had burrowed through the fascia and appeared as a raised "boil" on the skin surface. As the drained "boil" had not healed, a biopsy was made and a diagnosis was made of adeno-carcinoma of the bowel and correctly estimated as that of the appendix. This then is the only case on record where carcinoma of the appendix has been diagnosed before operation.

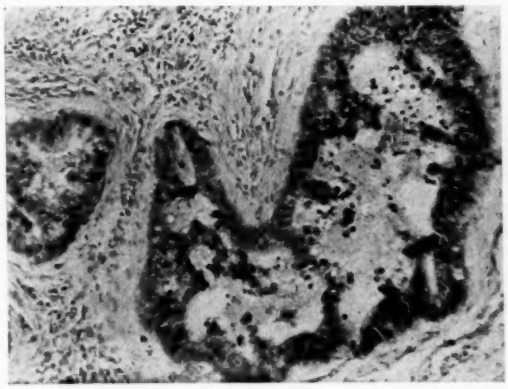

Fig. 2. Microscopic appearance of the gelatinous adenocarcinoma of the appendix.

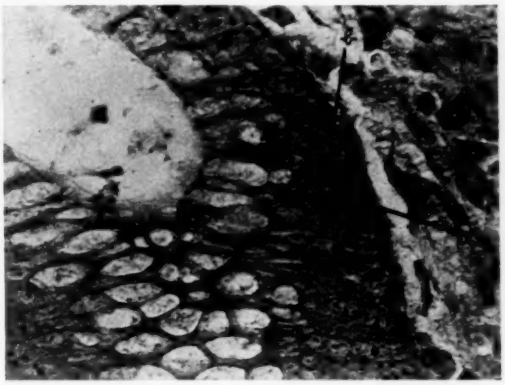

Fig. 3. Base of a Lieberkuhn crypt. (a) Argentaffin cell. (b) Its nucleus.

Hasegawa[2] states that the process of growth of the carcinoid is very slow and thinks most likely in the great majority of cases these tumors begin developing in early youth.

In an elaborate article Hasegawa establishes the relation of the carcinoid tumor to the argentaffin cells of the Lieberkühn crypts, and also demonstrates the metastatic possibility of the carcinoid tumor. He found that in the inflammatory process of the appendix, whether mild or acute, in which obliteration of the lumen takes place there is a process going on of amputation of the sympathetic nerve so that a neuroma of the sympathetic nerve develops, and speaks of it as resembling an amputation neuroma of peripheral nerves. In these cases Hasegawa could demonstrate in serial sections that the argentaffin cells separate themselves from the columnar cells of the Lieberkühn crypts and sink into the deeper parts where they are constantly found surrounding the sympathetic "amputation" neuroma. Scattered throughout these neuromata were found cells resembling ganglion cells but in them could be demonstrated the argentaffin granules.

So silver salts are taken up by the granules of: (1) the few scattered argentaffin cells found normally in between the goblet cells of Lieberkühn crypts; (2) the cells of the carcinoid tumor; and (3) the ganglion-like cells of the amputation neuroma.

Hasegawa developed the following staining technic: Small pieces of tissue of an appendix or of a carcinoid tumor are fixed in formalin, then thoroughly washed in running water. Now the tissue is placed in a 2 per cent solution of silver nitrate and incubated for twenty-four hours at 98.6. After a short period of washing in distilled water it is again placed in an incubator for twenty-four hours immersed in the following ammonium hydrate silver nitrate solution: To 10 c.c. of a 2 per cent solution of silver nitrate are added seven drops of a 10 per cent solution of either sodium or potassium hydrate and the resulting precipitate is dissolved by using dropwise, liquor ammonia. After a running water treatment the tissue is dehydrated, embedded in paraffin, sectioned thin (5 micron)

and mounted. It is to be noted that the outside sections are very black. Then follow the sections of proper impregnation and deeper insufficient silver will be found.

Argentaffin cells[4] are common in the crypts of Lieberkühn of the duodenum and appendix, infrequent in the jejunum, ileum and colon, and occasionally may be found in the stomach. Rarely are they found among the cells of the villi.

The argentaffin cell adheres closely to the basement membrane, is bottle shaped with the narrow part of the cell to the lumen. These cells may pass down through the limiting membrane of the lamin propria into the connective tissue. They are found singly or in groups and here they may become intimately connected with the nerves of the plexus of Meisner or form the carcinoid tumor.

There is nother special cell in the Lieberkühn crypt called the Paneth cell which is found only at the bottom of the crypt and only in the duodenum, jejunum and ileum. The Paneth cells have spherical nuclei and the cytoplasm at the lumen end has scattered large round secretory granules.

The argentaffin cells also have spherical nuclei, but have a fine compact mass of granules in the cytoplasm at the base. The granules of the Paneth cells respond to the chromaffin stains while the granules of the argentaffin cells respond to the silver stain.

The argentaffin cells make their first appearance in the fourth month of fetal life while the Paneth cells appear in the seventh month. The function of the granules of the argentaffin and the Paneth cells are not definitely known. Some histologists claim the Paneth cells are young goblet cells but if that were so we should also have the Paneth cell in the Lieberkühn crypts of the colon where they are definitely absent.

The interesting physiology of the intestinal glands and villi is that, besides producing digestive juices, there is a massive shedding of epithelium going on all the time. This requires a constant replacement, and the new cells develop in the bottom of the Lieberkühn crypts. So that the feces of a starving patient consist of the intestinal juices, mucus, bile and the desquamated epithelium.

Hasegawa quotes Masson who made this interesting observation: "Characteristically there are two types of cells in the carcinoid tumor, one having vacuoles containing lipoids resembling the adrenal cortex and the other type having chromaffin granules resembling the cells of the adrenal medulla. With these characteristic likenesses in mind Masson named the growth "endocrine tumors of the appendix."[4]

Huebschman is quoted by Hasegawa in the declaration that the origin of the carcinoid cell is either from the Paneth cell or the argentaffin cell of Schmidt-Ciaccio.

Krompecher, who also is frequently quoted by Hasegawa, draws comparisons between the basal cell of the skin and the occasional cell found among the columnar epithelial cells of the intestinal or Lieberkühn glands as being also basal cells. He calls the tumors forming from these cells "Basaliom."

Scott[6] records the age range from five to eighty-one years.

Vance[9] reports that carcinoid has been reported in the literature as becoming malignant nine times.

The incidence of primary carcinoma in all appendices examined is 0.35 per cent, as reported by Selinger.[7]

The incidence of primary malignant tumors of the appendix varies in different large clinics. In large clinics about 1 per cent of carcinomata of the bowel occur in the appendix. Of all primary malignancies of the appendix, 10 per cent are carcinoma and 90 per cent are carcinoid.

In 1936, Rosenblatt and Robertson[5] reported on 360 cases of primary carcinoma of the appendix. Rosenblatt, et al, report 0.2 to 0.5 per cent of all surgical appendices, as carcinomatous, while less than 0.5 per cent of all intestinal carcinomata are found primary in the appendix.

At St. Joseph's hospital, Saint Paul, from January 1, 1933, to September 1, 1939, the total number of appendices removed and studied were 3,692. Of this total number, our case is the only primary adenoma carcinoma of the appendix (0.027 per cent of all appendices studied). During this period there were eight cases of carcinoids reported or 0.216 per cent of all appendices studied.

Conclusions

1. Primary adenocarcinoma of the appendix is comparatively rare.

2. It is futile to attempt to make a preoperative diagnosis.

3. If a rupture of the appendix occurs, metastases will be present within a year.

4. The similarity and difference between the adenocarcinoma and the carcinoid tumor is emphasized.

5. Other names for carcinoid tumors are: (a) mucous membrane nevus; (b) endocrine tumor of the appendix; (c) basaliom.

6. A case of primary adenocarcinoma is reported.

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◆ CASE REPORTS ◆

THE TREATMENT OF MYASTHENIA GRAVIS WITH ORAL PROSTIGMINE*

Report of a Case in a Patient with Congenital Hemolytic Jaundice

DAN. L. FINK, M.D., and ARTHUR J. MOSS, M.D.

Minneapolis, Minnesota

THE earliest description of myasthenia gravis was presented by Dr. Willis in 1685.[26] He said of "The Palsy" that ". . . motion fails wholly in part or member, but is performed weakly. . . . There is a scarcity of spirits so that on first arising in the morning they move their arms this way and that . . . but by noon, the store of spirits being spent, they are scarcely able to move." He describes a case in which the tongue was involved so that after talking for a time, the patient became mute for an hour or two. More accurate and precise descriptions of this condition were presented by several German investigators in the latter part of the 19th century, and by Wilks in England in 1877.[25]

Since that time many have studied the etiology of myasthenia gravis. As a result, numerous theories have been propounded, placing the primary disturbance in the endocrine system, the nerves or the muscles.[12] Metabolic disturbances and chemical imbalance have been considered of significance.[1] The fact that thymomas are frequently found in cases of myasthenia gravis has led some investigators to believe that the underlying cause is of a neoplastic nature.[10]

The present status of the etiology is best presented by Pritchard, who found the nerve and muscle to be physiologically normal, but found that electrical stimulation of a nerve in myasthenia gravis gave a characteristic curve placing the defect at the myoneural junction.[16] He believes, together with many others, that acetylcholine plays an important rôle in the transmission of impulses across the myoneural junction. In myasthenia gravis, it is believed this chemical is diminished by the overactivity of the choline-esterase present in the blood.[6,15,16] Even this theory has been refuted by recent experimental work in Germany.[9]

Correspondingly, extracts of the ovaries, hypophysis and tuber cinereum have been used in the treatment of myasthenia gravis, but without success. Strychnine, adrenalin and parathormone have been tried; some have used calcium, presuming the mineral metabolism to be at fault.[12] Extirpation of thymic tumors has also been advocated as a therapeutic measure.[10] Ephedrine[7,14] was used with supposedly good results in some instances and more recently glycocoll has been advocated.[8] All these substances failed to control the disease to any great extent and reportedly good results in many instances were apparently due to spontaneous remissions of the condition.

In 1934, Walker presented her method of therapy.[24] In a letter to the editor of the *Lancet* she said that because myasthenia gravis had been thought to be due to a curare-like poisoning of the myoneural junction, she thought it would be well to try the effect of physostigmine, a partial antagonist of curare, in the treatment of the disease. Her attempt in a single case of myasthenia gravis gave gratifying results. Later, she reported on the successful use of prostigmine, a similar, though less toxic, drug.[13,23] The co-relationship of curare and prostigmine in myasthenia gravis was later demonstrated.[4]

The use of prostigmine by mouth was introduced in 1935 by Everts, who found that in addition to being more readily administered, its effect was more prolonged and with fewer untoward reactions.[8] Several investigators have felt that the use of prostigmine was

*From the University of Minnesota and Minneapolis General Hospital.

dangerous because of its toxic effects, and have advised against its oral administration until more is known of its fate in the gastro-intestinal tract.[2,11] However, the men who have used it the most, notably Viets and Schwab and their co-workers, feel that in myasthenia gravis the toxic effects are not a serious hazard.[10,20,21,22]

The drug to be administered by mouth is supplied in 15 mg. tablets and the dosage must be individualized to get the best therapeutic effect without injurious side effects.[8,18,22] Its action is apparently enhanced by the addition of ephedrine as was recently shown by Viets and Schwab in a study of forty-four cases, in 41 of which ephedrine increased the effect of the prostigmine.[22] Schafer used benzedrine as an adjunct.[17]

In the past twenty years the diagnosis of myasthenia gravis had been made but three times at the Minneapolis General Hospital. The first case was that of a patient who apparently had a spontaneous remission after symptomatic treatment only and has not been heard from since (thirteen years). The second patient expired before therapy could be instituted. The case presented here is the first to be treated with prostigmine at the Minneapolis General Hospital.

Case Report

Mrs. C. H., a thirty-four-year-old white woman, was admitted to the hospital on August 31, 1939, giving a history of progressive generalized weakness for three to four months and cough and fever for three days. For the preceding three months she had become progressively weaker. She seemed quite rested and was able to do her housework in the morning, but by evening she was "all tired out"; after walking a block she became so exhausted she had to stop and rest; after eating part of her meal, she had to use her hand to assist her jaw; and ptosis of her upper lids had become pronounced. For three days prior to admission, the patient had had fever with increasingly severe dyspnea, dysphagia, ptosis and cough, with inability to raise sputum.

Review of her past health revealed that the patient had congenital hemolytic jaundice, confirmed by laboratory studies; and that in January, 1938, after a severe, acute exacerbation of the jaundice, her spleen had been removed. She was seen in the Minneapolis General Hospital on two other occasions with findings of acute cholecystitis and cholelithiasis, but no mention of symptoms of myasthenia gravis was made until March, 1939, when it was noted only that "patient says she becomes very weak at the end of the day." In retrospect, the patient claimed to have had diplopea as early as November, 1938.

Physical examination revealed a critically ill patient almost moribund, very cyanotic and dyspneic with marked ptosis of both lids, dysphagia and dysarthria. Blood pressure was 120/70. A diagnosis of myasthenia gravis was made and at 2:40 A. M. 3 c.c. of 1:2000 solution of prostigmine methyl sulfate (1.5 mg.) and atrophine sulfate (gr. 1/150) was given intramuscularly. By 2:50 A. M. all the symptoms objectively and subjectively had either disappeared or had remarkably improved. She was able to cough and expectorate, to speak distinctly, to eat and drink, and her respirations became easier with disappearance of the cyanosis. The patient stated that she had not felt so strong for weeks.

Her temperature was 102° and there was evidence of consolidation in both lower lobes on physical examination and on x-ray study of the chest. No thymic enlargement could be demonstrated. Sulfapyridine therapy was started, the patient getting 4 gm. daily for five days; and she was placed in an oxygen tent for twenty-four hours. By the seventh day her temperature was normal and remained so.

The effects of the prostigmine lasted until 8:00 A. M., at which time the symptoms had recurred and the same dose was repeated with the same startling results. She was then started on prostigmine bromide by mouth augmented with ephedrine hydrochloride. The dosage was 15 mg. every 2 hours from 8:30 A. M. to 10:30 P. M. with ephedrine hydrochloride gr. 3/8 given at 7:30 A. M., 10:30 A. M., 4:30 P. M. and 8:30 P. M.

For the next few days it was necessary to augment the oral medication with the intramuscular drug on several occasions. By September 6, 1939, the signs and symptoms had almost entirely disappeared and the patient was up and about the ward. The dosage was then gradually reduced so that by September 21, 1939, she was taking but 30 mg. of prostigmine bromide daily together with four doses of ephedrine hydrocloride. However, on the dosage the patient developed diplopea, dysarthria and dysphagia; while on 3 tablets daily these symptoms disappeared again.

Laboratory studies revealed a normal urine, a hemoglobin level of 78 per cent, and white blood count of 37,500. Blood calcium was 11.5 mg. and phosphorus was 4.6 mg. Plasma chlorides were 276 mg. per 100 c.c. The blood Wassermann test was negative; the icterus index was 12. The average red blood cell diameter was 7.2 micra, and increased fragility of the red blood cells could be demonstrated. Urine chlorides were 2.04 gm./100 c.c. Sputum on admission revealed type 28 pneumococcus. Basal metabolic rate, electrocardiogram and spinal fluid were all normal.

The patient was discharged on September 23, 1939, asymptomatic, taking 15 mg. of prostigmine bromide at 10:30 A. M., 4:30 and 8:30 P. M. together with 1 capsule of ephedrine hydrochloride (gr. 3/8) each time. Since then she has been followed in the Out-Patient Department and when last seen, three months after admission, she had gained fifteen pounds in weight and was free from symptoms. On our suggestion she discontinued the drug on one occasion with prompt recurrence of the syndrome.

Conclusions

A case of myasthenia gravis is reported in which prostigmine bromide by mouth combined with ephedrine hydrochloride is being used effectively.

The case is further of interest because of the history of congenital hemolytic jaundice and splenectomy. The congenital hemolytic jaundice preceded the myasthenia gravis.

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HISTAMINASE IN THE TREATMENT OF URTICARIA OF PREGNANCY

J. S. BLUMENTHAL, M.D.

Minneapolis, Minnesota

THE use of histaminase in the treatment and prevention of allergic signs and symptoms has received a great deal of attention recently. The theory is that the release of histamine in the tissues is the immediate cause of the phenomena of allergy. The result of treatment as well as the theoretical basis for it has been reported with varying results in the literature. Best[1] showed that lung and other tissues when suspended in saline solution and incubated in the presence of toluene at 37 C caused the disappearance of natural or added histamine. Best and McHenry[2] also noted that there appeared to be a marked relation between histamine shock and anaphylactic shock. The substance which produced the disappearance of histamine is thermolabile and in 1930, Best and McHenry[3] suggested the name histaminase. They stated, however, "Since it has not been established that histamine is the causative agent in any pathological condition there would be no obvious clinical application of histaminase, even if it should be established that the abilty of an organism to inactivate histamine can be increased by administration of the enzyme." Laymon and Cummings[5] in a recent publication of a presentation before the Society for Investigative Dermatology came to the conclusion that histaminase is apparently of value in some cases of allergy, though they could not attest to any specificity. Dr. C. W. Laymon, in a personal communication, states that he is himself convinced that histaminase is very useful in many cases of urticaria where other methods have failed. Miller and Piness,[6] on the other hand, in a later publication, came to the conclusion after treating forty-two allergic patients including twenty-eight with urticaria, that histaminase failed to give unequivocal evidence that this enzyme was responsible for the relief or prevention of any signs or symptoms of which the patients complained.

Karady and Browne[4] in 1939, however, recorded a successful attempt to neutralize the action of histamine in the guinea pig by using histaminase (Winthrop).

In the case presented here, the histaminase used was that manufactured by the Winthrop Chemical Company and called by them "Torantil." The product is dispensed in tablets and is said to be derived from hog's kidney, the unit of activity being "the amount which will inactivate one mg. of histamine hydrochloride during incubation at 37.5 C. for twenty-four hours." These tablets containing five units each are to be administered before meals and with one glass of water. The usual dose recommended is from five to ten units three to four times daily.

The patient presented here is a white woman, thirty-one years old, whose last menstrual period was July 26, 1939, and who therefore was due to deliver about May 2, 1940. She is a primipara, 5 feet 4 inches in height and her usual weight has been 112 pounds. She gave no history of any previous skin abnormality. She has a history of a mild degree of hyperthyroidism which had been treated by x-ray with apparently good results so that she has no symptoms referrable to her goiter at present. Her basal metabolism has been normal for several years. She had taken iodine occasionally because she said it made her feel better and she had continued to take a few drops of Lugol's solution at times, throughout her pregnancy. There is no past history of allergy of any type and she had never had any skin lesions. Her family history is negative except for a history of a twin pregnancy by her mother's sister.

The patient's physical condition when first seen was good and that of a normal pregnancy although the possibility of twins was suggested due to the large size of the uterus. Her weight was 133 pounds, blood pressure was 120/80, her pulse rate 90, fetal heart rate 140. Urine was normal. Hgb. 90, r.b.c. 4,200,000, w.b.c. 7,000, Wassermann negative. Measurements were normal.

The pregnancy was uneventful in all respects until February 17, 1940, when a small area of redness developed about the vulva with a very annoying itch. The itching increased very rapidly and the urticaria spread until it covered the whole body. Elimination diets, stopping of iodine, starch baths, ultraviolet ray, anti pruritics of any and all kinds and intravenous sodium thiosulphate were tried and to no avail. Sedatives, in-

(Continued on page 824)

DISEASES OF THE DAKOTA INDIANS*

By Thomas S. Williamson, M.D.

(Continued from the October issue.)

During those years I saw several cases of croup, one of which proved fatal; but I do not remember to have seen a well marked case of it among Indians since. Influenza was endemic every winter, generally beginning very soon after those who had been away hunting and living on animal food returned and began to mingle with those who remained in the neighborhood and subsisted almost entirely on corn and potatoes. As there was always much feasting at such times, I at first attributed it entirely to over eating, and still think this may have had something of a causative nature, but those who made no change in their diet at such times did not escape. I observed that all persons connected with the mission, i.e., white persons from the States, were exempt from this influenza the first winter and generally had it lighter than others the second; after which they suffered about the same as the others. I am not aware that the disease ever proved directly fatal there, but it induced other diseases that did.

Except from this influenza, which seldom prevailed for more than a month at a time, the Indians, notwithstanding their scant clothing, suffered much less from what are usually called colds than white people, and this I think was owing to their scant diet.

Almost every year of my residence in Minnesota I have seen several cases of paludal disease, but during the first ten years of my residence at Lac qui Parle I saw only one such in that region which proved fatal. This, like most of the others, had its origin in the swamps of the lower Minnesota and Mississippi. Little Crow, afterwards so infamous, visited Lac qui Parle in the spring and took with him on his return to the village below Saint Paul (i.e., where it now stands) a young woman who returned to her parents during the summer and soon after was taken down with remittant fever. I was called to see her and had no doubts as to my ability to administer such medicine as would, with the blessing of God, remove the disease—but I could not conscientiously do this while the conjurers were pow wowing over her; and a young man to whom her younger sister had been given as a fee for doing this refused to give way to me—so she died, as I expected she would under such treatment.

The summers of 1845 and 1846 were warmer than any for a long time previous, in Minnesota, and hence there was a great increase in such diseases as are caused by decaying vegetable matter. During the autumn of 1845 many of the children suffered from cholera infantum, dysentery and fever; and during the following winter several aged persons died of intermittent fever; these I suppose might have recovered under proper treatment, but I had little quinine and it was impossible to get more; they also lacked animal food and good nursing, as their friends were, most of them, absent on their fall hunt.

In the autumn of 1846 there was more sickness and more deaths from the same cause, but how many I cannot say as I saw but a small part of the sick; for during this same autumn at the request of the Indians of Little Crow's village and

their agent I removed from Lac qui Parle to their village, a few miles below Saint Pauls. I have been thus particular as to the Indians at Lac qui Parle, for two reasons: first, notwithstanding the great lapse of time I have a much clearer remembrance of their condition, sicknesses and deaths, than at a later period; second, I had an opportunity of studying them at that time in their primitive condition, living without bread, pork or salt, such as has occurred to few physicians, and is not likely ever to occur again.

Notwithstanding the prevalence of scrofula and much suffering from want of food and clothing, if it had not been for violent deaths they would have increased in numbers nearly as fast as our own white population. I saw at times many of the Indians of the plains who subsisted at most times entirely on buffalo meat, they generally appeared real healthy with the exception of sore eyes. Scrofula is certainly less prevalent among them than among such as subsist almost exclusively on vegetable food, and until the buffalo began to fail they were increasing rapidly, in spite of the occasional ravages of small pox and measles.

I will here mention some of the causes of phthisis among the Dakotas; I have already mentioned scrofula, and alluded to the principal cause of that, hence it is not necessary to say more about that disease. Next is over exertion in order to obtain a subsistence. It has been often said and most people believe that the Indians lead a very easy, lazy life; but this is far from true. From the time I came to Minnesota until twenty-one years ago the Indians of this State depended for a subsistence chiefly on hunting; though most of them raised some corn, which, at certain seasons of the year, was the principal, and sometimes the only, food. Hunting may be very pleasant to those men who engage in it only for recreation, but it is hard and unpleasant as work. Near Lac qui Parle there were few or no deer and the principal animal food was wild fowl, which were obtained chiefly in the fall and spring when the water was little above the freezing point, and the hunter after walking from one to twenty miles to a sheet of water where ducks were plenty waded in among the tall grass and stood in water often knee- and sometimes waist-deep for hours to get a fair chance at a good flock. His ammunition cost him so much he could not afford to shoot at fowls on the wing or at long range, for each charge of his gun cost him at least as much as the amount for which one duck would sell. If he were so fortunate as to shoot his game without wading, still in most cases in order to get it he must wade into deep water and sometimes swim; and when it was secured he frequently had to carry it ten or fifteen miles over a bleak prairie, with all his clothes, except his blanket, wet, and often stiff with frost. A few deer were killed during the summer within ten to twenty miles of the villages on the Mississippi and lower Minnesota, but each of them usually cost many days' walking through thickets. The principal deer hunt was in autumn and early winter, when from fifty to one hundred families went together, for smaller parties would be cut off by their enemies. In such expeditions, besides their clothing, bedding, wooden dishes, each woman's workbag, and children unable to walk, it was necessary to carry for each family a skin tent weighing from forty to one hundred pounds, an iron kettle for boiling their food, an ax, a hoe for leveling the ground where the tent is to stand, and some food, as not unfrequently no game was killed for a day or two. The country to be hunted over and the place for the next encampment having been determined on the night before, as soon as it is light hunters start, each carrying his gun, ammunition, apparatus for making fire, knife and a small hatchet. The mistress of the family packs and binds in bundles the tent and its contents assigning to each member of the family his portion of what is to be carried, taking

the heaviest burden herself, unless the family owns a horse, in which case that takes the largest share, and she the next, each still carrying something. Many families had no horse and in those the burden of each woman in middle life did not average less than 100 pounds, and sometimes much more; this was usually carried from four to ten miles a day, averaging about six. Sometimes when a wide prairie without a camping place was reached, or a scare from enemies occurred, their loads were carried from twenty to thirty miles in a day and part of the night, and this through woods or the long grass of the prairies.

You will not think it strange that such taxing of the muscles and nerves should bring on phthisis, sometimes by mere exhaustion, but oftener by causing hemorrhage from the lungs.

It was supposed and said by persons casually passing through the Indian country that the men had a much easier time than the women, and it is true that, as is the case among civilized people who labor for subsistence, the men had more time to rest than their wives, that is, were oftener idle. In hunting, however, the toil of the men was more exhausting than that of the women. The deer hunter was often unsuccessful after having run forty or fifty miles without a road or having tasted food, while if successful in killing a deer it was generally after chasing it many long miles; and, being obliged to bring in the whole carcass, his load was frequently much heavier and he had farther to carry it than any of the women.

Cases of hemoptysis were more frequent among men and more of them died from diseased lungs than the women. The life of those who subsist on buffalo was very different as they had many horses, but among them, too, the young men often suffered severely, first in finding the buffalo, and next in killing them. As their clothing is made almost entirely of buffalo skin, which is too stiff to allow of free motion of the parts it covers, they were accustomed to chase the buffalo on horseback, even in the coldest weather of winter, with their arms and all the body above the belt entirely naked. These dwellers on the prairie when near buffalo were generally fine and healthy, but when they could find none of these animals suffered even more from starvation than the other Dakotas. This suffering from lack of sufficient food is another cause of phthisis.

Having assigned the causes of the prevalence of this disease among the Indians, I might here close my already long communication, but will mention some other facts which will be interesting to you.

As above mentioned, I moved to Kaposia, an Indian village six miles by the river below Saint Pauls, in the autumn of 1846. During the winter I was called to see several genuine cases of ague, and a much larger number of abnormal cases of paludal disease; and here I will state that this disease which used to be called bilious fever is much less regular in form in Minnesota than it was in Ohio when I practiced there. Usually there is much less of what is called fever, that is of heat and arterial action and often the chill or sweating stage is scarcely perceptible; so that, though readily recognizable by persons familiar with it, even regular physicians who know it merely from description in books are very liable to mistake it for some other disease. Since I have ceased to practice medicine I have seen several instances of this, two of which proved fatal, as appeared to me, in consequence of this mistake. The summer of 1847 also was unusually warm and cases of paludal disease were numerous, both among whites and Indians in the country near Fort Snelling. The Dakotas suffered most because, like our British ancestors, for security against their enemies they had made their villages in the midst of swamps, and also because they lacked food. Government usually

gave them their flour and pork in the month of June, but that year it was withheld for a month or two, and many of them died in consequence. In health they could have subsisted on roots, chiefly of sagittaria, obtained from swamps, and on wild ducks, pigeons and fish. But the stomach of the sick rejected fish and pigeons; and it often happened that when a man, after wading in swamps for hours, returned with ducks, or a woman with roots, he or she would fall down in a chill. The food brought home was speedily prepared, but the bringer, in a chill or fever, could take no portion of it then, and before the fever was gone and appetite had returned some hungry child had eaten all the food.

I suppose a majority of the Indians were more or less affected with the disease, and about one in thirty died of it. Many more would have died but for food given them by their white neighbors, and probably four-fifths of those that died might have been saved by proper food and medicine. Subsequent summers were cooler, more healthy sites were selected for some of the villages the next year, and there was less of the disease, and subsequently but little.

Some years later these lower Sioux, as they were called, suffered severely from whooping cough in th spring and early summer. To the children who were brought to me soon after the attack, I gave small doses of tartar emetic for the first two weeks, keeping their stomachs a little irritable so that when the cough came on severely they would vomit a little. Later in the disease when they became feverish or the stomach so irritable as to reject necessary food, I gave laxatives, and of those thus treated I think not one died. At other villages the mortality was considerable. I was taken to see the sick after more than twenty had died, out of a population of about 400. On examining the little sufferers it was manifest they were perishing for lack of nutriment; and on inquiry I found that the whole village had for some time been subsisting solely on fish, and whenever a paroxysm of coughing came on they vomited what had been eaten. Medicine could do but little for them, so I told the chief to call together his principal men, told them all to look at the sick children and gave them a severe scolding for letting the children starve. When they pleaded poverty as an excuse I told them to send all the young men immediately to hunt deer, and for the older ones to go to their traders and get corn, flour and sugar for the children. They did as I directed and very few died afterwards.

Similar treatment was less successful when the disease prevailed in winter, because it was impossible to keep the patient comfortably warm.

DAKOTA MEDICINE*

By Rev. Thomas S. Williamson

AMONG the Dakotas, as among other heathen races, the offices of physician and priest were, for the most part, united in the same person. This being the case, it is not strange that their pathology should be shaped by their ideas of the spiritual world. Supposing every object, artificial as well as natural, to be the habitation of a spirit capable of hurting or helping them, and that all diseases were caused by some one or more of these spirits taking possession of a part or the whole of the body of the patient, to determine the name and nature of the spirit causing the trouble was regarded as the first business of the physician or *con-*

*From "Tah-Koo Wah-Kan; or The Gospel Among the Dakotas," by Stephen R. Riggs, 1869, p. 435.

jurer, as we usually call the medicine man of the aborigines of our country. This he attempted, not only by observing the symptoms, but by incantations addressed to the spirit or spirits which were the special objects of his worship, and expected on that account to befriend him.

The second business of the medicine man was to drive out the intruding spirit. This was attempted by all kinds of horrid noises and gestures, not omitting to call upon his god or gods for assistance. He also, in most cases, applied his mouth to the skin of the patient near the diseased part, and, after sucking for some time with all his might, would put his mouth in water kept in a vessel usually made of bark for that purpose, and blow into the water the phlegm, mucus, or blood, which he pretended to have drawn from the seat of the disease through the skin; but in most cases they doubtless came from his own mouth or throat. In many cases he would also introduce through his mouth into the water a small pebble or shell, which he would show as evidence that he had extracted the offending cause of the disease through the skin. As in this work, by shrieking, screaming, groaning, shaking his rattle, stamping and other threatening motions, the conjurer exerted all his strength, it was customary, even in cold weather, to divest himself of all clothing except his breechcloth, moccasins, and leggins; and the latter, instead of being bound about his legs, hung trailing from his ankles.

When medicines are administered, their efficacy is attributed to the god or spirit residing in the plant, rather than to any mechanical, chemical, or other power inherent in the medicine itself.

The number of practitioners is large, in some villages not less than one man in ten and one woman in thirty. Among them are to be found some of the shrewest, strongest intellects, and a very large proportion of those who are only one step above idiots. A proper idiot I have never met among Indians, probably because such are left to perish in infancy; but the proportion of those who make an approach to idiocy is much greater among savages than among civilized people; and a large part of these are applied to in cases of sickness. Many of the conjurers are known as such by that peculiar cast of countenance which belongs to the spiritist of civilized nations; others have countenances strongly indicative of cunning and deceit. I have met two or three men among them of good, plain, common sense, and open, honest countenances. The most eminent of these, when on a certain occasion he came to me for medicine for a disease brought on by over exertion and exposure in the practice of their orgies, on being charged with deceiving his people, acknowledged the charge, and excused himself on the ground of his poverty and his need of the promised reward, and the number and urgency of those who applied to him to show them by his incantations how and when they might find and destroy their enemies.

There are a few individuals who give medicines without conjuring or invoking the aid of spirits. I have known one such who attained a deservedly high reputation among his own people and was sometimes called to practice among the whites. With intellectual and perceptive faculties strongly developed, and such knowledge of the powers of roots as is attainable among his people, naturally polite, and very observant of all those little things which contribute to the comfort of a patient, I have often thought he might have acquired fame and money among civilized men, if he had not justly preferred to live among his own people and do what he could for them.

The medicines used by the Dakotas were mostly roots, and their name for a physician, Pay-zhe-hoo-ta-we-chashta, Herb-root-man. They, however, used not only other parts of plants, but sometimes animal and mineral substances. In

general their medicines were all secret, the knowledge of them concealed as far as possible from each other, except in cases where large fees were paid by such as were not likely to become rivals in the same community, or where an individual not expecting to practice his profession much longer, owing to age or infirmity, might communicate to some descendant or other near relative the knowledge which he possessed. This disposition to conceal their knowledge, so prevalent among quacks, not less than the want of books, prevented the transmission and accumulation of knowledge; so that if any important discoveries were made, they were liable to perish with the discoverer.

With such false pathology and such hindrances to the transmission of knowledge, we could not expect that the sick would be much benefited by the medicine men. So far as I have had an opportunity of observing their practice in cases of fever and some other diseases, with very few exceptions, they did more harm than good. But I know of no way of accounting for the high value set on their services without supposing that, in many cases, they gave relief. My observation inclines me to think that this occurs especially in wounds and pains from local inflammation more frequently than we should expect from what has been said above.

The perceptive faculties among the Dakotas are far more acute than among civilized men. The successful medicine man, like the successful warrior, attains to the highest honor, and is more sure of getting property largely, than either the warrior or hunter; and thus his faculties are stimulated to the highest degree of activity; and having usually only one patient at a time he can observe more closely the effect of the medicine he uses. Some of this class are excellent nurses of the sick, and most attentive to all those thousand little things so conducive to the comfort of their patients. The family is removed from the tent, or the patient and tent from them; and the ground which constitutes the floor of the tent is carpeted, in summer, with ferns or other soft herbage, and scented with aromatic herbs, and, in seasons of the year when these cannot be obtained, with the best substitutes for them accessible. Some of the medicine men watch their patients very thoroughly, and to prevent noise exclude children, and in many cases women too, unless the nearest relatives.

They practice blood-letting, which with them is an operation generally performed with a sharp flint which serves very well for scarifying. When a vein is to be cut, the flint is made fast in a stick, which serves as a handle, and, like a fleam used in bleeding horses, it is driven in with a stroke. As this is a difficult operation, few attempt it; but local blood-letting is very common, the operator usually drawing the blood directly into his own mouth. Some, however, use a horn, or a part of one, applying the large end to the surface whence the blood is to be drawn, and taking the smaller end, which is perforated, into the mouth.

They attribute many of their diseases to bile, or "yellow water," as they call it, and for its removal use emetics, purgatives, and clysters. Most of them, however, freely acknowledge that none of the native remedies for certainty and safety equals those used for purging and puking by civilized men, since to secure vomiting the Dakota doctor is commonly obliged to tickle the throat with a feather.

For purging, the Dakotas who lived near the Mississippi, in common with their eastern neighbors, the Ojibwas, relied chiefly on the root of a tall and very handsome species of euphorbium which abounds on the prairies between Saint Paul and Red Wing. Coarsely powdered it is administered in small quantities, and it operates quickly and often severely. They say it is safe if used dry and the patient abstains entirely from drink until the operation is over, but very dangerous

if the patient drinks freely soon after taking it. The Ojibwa chief, Firm Earth, the predecessor and older brother of the first Hole-in-the-Day, who died near Fort Snelling in 1843, was said by his companions to have come to his end by drinking freely after taking a dose of this root. He died in a few minutes after walking about, and apparently from the effects of acrid poison. The plant is not found on the Upper Minnesota or farther west, and consequently I have had but few opportunities of witnessing its effects. The Indians who have not access to it, use various other plants, none of which appears to have much efficacy. The same may be said of those used as diuretics.

To promote appetite, especially after fevers, they used cranberries, preferring for this purpose the fruit of the tree cranberry, and sometimes substituted bitter aromatic roots.

Sweating in a small tent, over heated stones, was a frequent and perhaps their most efficacious remedy for the removal of disease. It was resorted to not only in cases of pain and sickness, but as an antidote or purifier by those who had killed any person, or otherwise contracted ceremonial uncleanness. The process is described elsewhere.

Contrary to what might be expected, many of them use anesthetics. I have frequently heard of persons reviving after being apparently dead, and seen several such who appeared very much as is common with those who are recovering from the effects of an excessive dose of opium, who nevertheless, I have good reason to believe, had not taken it. In consequence of the conjurers interfering with some of my own patients, I had an opportunity of observing one or two in the state of anesthesia. It is caused by making the person inhale the fumes of calamus roots, and some other substances, burnt on coals. Whether the effect is produced by the calamus or some of the other ingredients, or by the combination, I am unable to say. This state is sometimes produced for the purpose of allaying that extreme restlessness which attends some diseases, but chiefly, I suppose, that the practitioner may have the credit of restoring a dead person to life.

The Dakotas were far more successful in the practice of surgery than medicine. Their constant practice in cutting up animals slain in the chase, made many of them well acquainted with comparative anatomy. Yet their ignorance about the circulation of the blood caused them to lose patients, who might have been saved by a judicious use of bandages, or the entire removal of them. They use tents to keep open deep wounds or abscesses, and prepare good ones for the purpose from the inner bark of the slippery elm. They sometimes apply wet dressings to wounds or sores, and cover them with green leaves, either fresh or boiled; but not possessing the materials for making poultices, plasters, or cerates, they generally endeavor to dry up and scab over wounds and running sores. Sometimes they cover the raw surface with a paste made by chewing certain roots, bark, or leaves. At other times the medicine is reduced to a powder and dusted over the sore. A variety of substances are used for this purpose, among which none is more highly esteemed than the root of the Asclepias tuberosa, a species of milkweed. This is also sometimes given internally.

To reduce swellings, especially those arising from sprains or bruises, they apply various stimulating vegetables, including tobacco. Among these they most value what they call *blue root*, a species of pyrethrum very common in the prairies of Minnesota and Dakota.

A few of them are skilful in the treatment of burns, but this is not generally the case. Commonly they apply oil or grease of some kind, when it is to be ob-

tained, and carefully conceal the knowledge of the other remedies they use. I once saw an excellent effect produced on a very extensive and severe burn, by covering the entire raw surface with the inner bark of the yellow, or, as it is called in Minnesota, the Norway pine. The bark had been shaved thin and made soft by beating it, and the inner mucilaginous surface applied, which allayed the pain and inflammation. I do not think any of the medicines of the shops could have a better effect.

When suffering from disease they will, with few exceptions, gladly avail themselves of the services and medicines of regularly educated physicians, and the conjurers not less willingly than others; although they endeavor to prevent others from doing so, claiming that their medicines are superior to the white man's.

FREE MEDICAL CARE

The man in the street may not understand how political medicine would lower the quality of medical care he receives but he certainly can understand taxes. And when he is told that the cost of such a program would amount to between 10 per cent and 15 per cent of the national payroll he can begin to see the joker in back of this promise of "free medical care."

During a recent session of the New York State Legislature, two bills were introduced which give us an idea of what we may expect if our busy propagandists succeed in foisting this scheme upon an American public deceived by false promises and unacquainted with the true facts.

These bills were alike in all essential details; both bills would have vested the control of medicine in a group composed predominately of laymen and entirely under political control. The medical representation provided would have been such a small minority as to have negligible power and although the bills provided for a professional advisory board, such a board would have had purely advisory powers with absolutely no authority to enforce its recommendations.

Of course nothing in this world is free and in the case of compulsory health insurance, the staggering bill would have to be met by the very people whom the bill is supposed to help. Although workers earning less than $20 a week were promised complete medical care for only $10 a year, the resulting deficit would have been made up by taxes on workers earning not much more. For example, salaries between $20 and $40 a week would be taxed up to $41 a year; between $40 and $60, up to $92 a year.

Even this would not be an intolerable load if it represented the entire cost, but the bills provided for a heavy tax upon the employers, which tax would of course have to be passed on to the workingmen either in the form of lower salary or in the form of higher prices for the commodities they manufactured, and then the deficit still remaining would be made up by general taxation, either direct or indirect, and everyone who is capable of any thinking at all understands that all taxes are ultimately paid by the man who works for a living.

—*Nassau Medical News.*

President's Letter

OUR GREAT TASK

THE American method of practicing medicine has developed the greatest advances in medical science and knowledge and has brought these to a larger proportion of the entire country's population than ever before in history. Never before has the whole population of any country received such efficient medical care as this country now enjoys. All the recent discoveries, inventions, and new developments that can aid medical care are being utilized; in addition to this, continuous experiments are being conducted in the endeavor to discover better methods of fighting disease.

During the past quarter of a century changes in every phase of life and knowledge have taken place; these changes have been slow enough to be regarded as an evolution and people have been able to accustom themselves to the new conditions without too great difficulty. But with these changes new ideas have been disseminated which are revolutionary in character and which if put into effect would completely change the manner of living of entire groups of our population. Experimenters, dreamers, and radicals, each with his peculiar ideals and theories, offer their ideas in alluring and enticing style and each would revolutionize the world. Among these theories Compulsory Health Insurance under government control, or Socialized Medicine, as it is often called, has been presented, in the rosiest and most attractive manner possible by large and powerful groups. Those who advocate it are either misinformed, ignorant, or actuated by selfish and ulterior motives. They would tear down and utterly destroy our established American system of medical practice and substitute for it the European system.

Should such a change be accomplished, not only will progress in medical science and knowledge be greatly retarded, not only will the initiative and ambition of the physician to advance in skill and knowledge be removed, but the status of the physician will be greatly lowered, his work reduced to drudgery, and his chosen profession will offer him but a bare living. Worse than that his opportunity to be of the greatest service to his patients will be curtailed. All of these things have been said before; but they are so vital to the public welfare that they must be repeated.

Since time immemorial we have been taught that the physician should not enter into the field of politics, that medicine and politics cannot mix, that the bickerings of political life will ruin the practice and usefulness of any doctor, however learned and clever. But times and conditions have changed. One need but consider the proposed bills introduced into our various legislatures and in Congress, or read the speeches and addresses of men high in legislative and executive positions in our national government to realize that this threat is serious and imminent. The radio broadcasts sent out in the past two or three months by the Department of Interior in Washington show what is going on under Mr. Ickes' direction. With but little regard for truth and no thought of how impossible or wasteful their schemes would be, they would have the people believe that all the ills of humanity could be cured by political control of medical care.

Unless we physicians enlighten the people, politicians will soon force government control of medical care upon us. It is high time for us to act. We can no longer remain an inarticulate group. However distasteful and no matter how difficult it may be for us as individuals to enter into this struggle, we must each take an active and energetic part or we shall all lose. We have allowed ourselves to remain in obscurity too long; it is time for us, now, to express ourselves and to do it forcibly and effectively. We must take an active part in public affairs and we must let people know that medical matters are vital and important to them. If we wish to retain our American way of practicing medicine we must let the public know what the European system is like, what its results are and what they can expect from such a system. Every physician has friends and patients who trust him and who believe in his honesty. He must talk frankly to these people. The time has come when the medical profession must speak, not only in its own defense, but in the defense of the people who are easily mislead to their own hurt. We should do this, not by argument or vituperation, but by calm statements of fact. As Dr. Andrew H. Smith told the New York Academy of Medicine: "The world has a right to know, and it is our duty to tell, just what progress we are making day by day, the steps by which results are obtained, the difficulties we meet, the uncertainties still to be cleared up, the problems which are pressing for solution." If we can gain the confidence of the public and help them to understand what it would mean to bring the European system to this country there would be no need for worry. To bring about this understanding is our great task.

BERTRAM S. ADAMS, M.D.
President, Minnesota State Medical Association

EDITORIAL

MINNESOTA MEDICINE

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BUSINESS MANAGER
J. R. BRUCE

Volume 23 NOVEMBER, 1940 Number 11

HISTORICAL HIGHLIGHTS ON APPENDICITIS

AS Treves has well remarked, discussions on the question of priority constitute the most pitiable and petty items in the literature of medicine. Any claim to priority in medicine or surgery rests not upon the date of performance but upon the date of publication (Kelly). However, the credit for a new procedure should go not to the person who merely first did it or wrote about it, but to the one who "put the idea across."

It seems incredible that the true nature of a lesion so obvious to us now as appendicitis should have been unrecognized for so long. In 1753 Heister first demonstrated at necropsy lesions occurring in the appendix. From the time of Mestivier of France, who in 1759 first operated upon an appendiceal abscess, until Fitz's paper in 1886, there were occasional reports on appendicitis in the literature, studied with amazing detail and skill, but the observers failed to trace the true origin of the disease. In 1827 Mélier, a French physician, presented a clinical description of appendicitis. However, Dupuytren, the leading French surgeon of the day, disagreed with some of Mélier's statements and held him up to ridicule. Mélier retracted his statements and, as a result, advancement in the knowledge of appendicitis was held back for 50 years (Collins). In 1880, Goldbeck and his school advanced the theory that the primary disease lay in the cecum and not the appendix. They advanced the terms "pertyphilitis, epityphilitis and endotyphilitis," which confused rather than clarified the situation. While Addison, Bright and Volz described the typical lesion in appendicitis, the credit for recognizing the true pathological nature of this disease belongs to Fitz (1886) who also gave the disease its name, appendicitis.

The surgery of appendicitis dates back about a century. Excellent reviews have been given by Collins and Kelly. According to Kelly the earliest efforts of the surgeon in the treatment of this disease consisted in opening a few abscesses and then gradually making the the incision before the detection of fluctuation (Hancock, 1848; Willard Parker, 1867). With the dawn of antiseptic surgery, a few surgeons ventured to open the peritoneum, to straighten out a kink of the appendix (Treves), to trim off the edges of a fistula in the appendix (Sands), and at last to remove the entire organ (Krönlein, Morton, Sands). To Thomas G. Morton of Philadelphia belongs the credit for the first appendectomy, deliberately undertaken with the diagonsis of acute appendicitis. In stressing the need for the early diagnosis and treatment of this disease, the names of Fitz, Price, Morton, Fowler, McBurney, Richardson, Morris, Murphy, Ochsner and Deaver deserve mention. To McBurney more than to any other surgeon belongs the cred-

it for crystallizing our ideas regarding the treatment of this disease. His paper entitled "Experiences with Early Operative Interference in Cases of Disease of the Vermiform Appendix", published in the New York Medical Journal for December 21, 1889, is a classic in the treatment of acute appendicitis. The subsequent years have not brought any important changes in his ideas of treatment.

It is interesting to note the cause of death of some of the investigators of the problem of appendicitis. Fowler died from peritonitis following a ruptured appendix. McDowell, who did the first successful ovariectomy in this country, also died from this disease. Price and Treves died of peritonitis; Fitz succumbed following an operation for chronic gastric ulcer. The etiology of the peritonitis in the cases of Price and Treves is not clear, although Price is said to have had an infection involving the retroperitoneal lymph nodes. Parker, Murphy, Ochsner and McBurney died from various forms of cardiovascular disease, the first from cerebral hemorrhage and the latter three from coronary disease. Deaver died of an obscure anemia, the exact nature of which was not determined even at autopsy. T. G. Morton died of cholera. It is noteworthy that both his brother and a son died of ruptured appendicitis, upon both of whom Morton had urged in vain the attending surgeons to operate.

It may seem somewhat surprising that the mortality of appendicitis is higher by occupation among physicians and surgeons than it is in the population at large. Whitney found that, from the mortality statistics of the United States census, the death rate for gainfully employed men (whole group, between fifteen and sixty-four years) was 20.7 per 100,000. For physicians and surgeons the rate was 22.1. Similarly, Alvarez found that medical men delay longer than lay people in seeking treatment for cancer of the stomach.

At present, the problem of appendicitis is not one of lack of scientific knowledge, but rather one of applying that knowledge effectively. Physicians would do well to apply that knowledge to themselves as well as to their patients.

CHARLES E. REA, M. D.

A SAFE SAFETY PIN

THIS product, the result of the inventive genius of Dr. Woodard Colby of Saint Paul, is now being manufactured and will shortly be available for general distribution. For those who have had the opportunity to use this pin, its merits are most appealing. Its construction is simple and most ingenius. The outstanding feature is the manner in which the point is concealed, making it impossible to cause injury to infants in the ordinary use to which it is put and virtually harmless if swallowed. It is to be manufactured by the Minnesota Homecrafters, Inc., under a plan sponsored by the Minnesota Department of Education and the United States Government. The pin has received the unqualified endorsement of the Ramsey County Medical Society and the St. Louis County Medical Society and of many leading physicians, among them Dr. Bela Schick of New York, Dr. Walter Ramsey and Dr. H. P. Ritchie of Saint Paul and many others.

The plan of manufacture and distribution as at present undertaken is two-fold. First, it will place a much desired article on the open market and, second, it will provide gainful employment for a goodly number of handicapped individuals. That these workers are by no means unskilled is evidenced by the fact that out of seventy-five useful articles exhibited at the Minnesota State Fair last month prizes were awarded to sixty-three. At the Tri-State Fair they received prizes for twelve from a total of fifteen shown, at the Proctor Fair ten out of twelve and at the Hibbing Fair every one of eight articles exhibited received awards.

Miss Marion Medd, Supervisor of this Homecrafters' project, informs us that a teacher provided through the Works Projects Administration visits the participating persons once a week and instructs those capable of skilled craftsmanship in the production of wood and/or metal articles. These articles are such that they can be sold on their own merit entirely and not on a sentimental appeal. Miss Medd is of the opinion that the Colby Clasp is ideally suited to this purpose because it can be completed in the home and will furnish work and income to this deserving group.

JAMES TRENT CHRISTISON, M.D.

MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
W. F. Braasch, M.D., Chairman

THE BLIND GODDESS

These are truly hectic days. Never has our American life been involved in such confusion and uncertainty as confronts us today. In the midst of turmoil the medical profession alone goes on its way, carrying on its mission of combating disease in any form which presents itself, as it has done in ages past. Conformists when necessary, but never obstructionists. So now, as emergencies arise, we change our methods to support our country's needs, making any necessary sacrifice without hesitation. All we ask is to conduct our healing art as we know it should be done and without interference from those who are not one of us. And now our shield and protection, the American Medical Association, in the midst of its efforts to support the nation's defense, is assailed by the Federal Department of Justice for daring to defy its plans for political control of medicine.

Trial Postponed

Suddenly the political element reveals itself, announcing that the trial is postponed indefinitely. And so, possibly in response to the ringing editorial which appeared in the last issue of the *Journal of the American Medical Association* and which was broadcast in the public press, the ban was suddenly lifted, at least until after election.

One would think that the terrific cost entailed in our nation's defense would stop such impractical schemes, but such is not the case. How stealthily the campaign to socialize medicine proceeds is shown by a recent attempt, under the guise of defense, to put the medical care of five million young men in the deferred list under control of federal health agencies. We, in the ranks of the Minnesota State Medical Association, can take pride in being the first to voice our opposition to this insidious plan which, if carried out, would eventually suppress the private practice of medicine.

Sword May Be Sharper

It is no wonder that the members of the American medical profession are intensely interested in the forthcoming election and are vigorously supporting those who uphold their principles. While the Damoclean sword has been temporarily sheated, it may be sharper than ever after election. In the meantime, it behooves every one of us to use every ounce of influence with all of those with whom we come in contact and to do what we can to protect the public welfare and the future of medicine.

MINNESOTA FIRST

The Council of the Minnesota State Medical Association is the first among responsible, executive bodies of state medical associations to go on record in opposition to free medical service for the rehabilitation of draft registrants rejected for physical defects. At the same time (as noted elsewhere in these columns) the Council went on record against the offer of Wassermann tests for all Class 1 registrants and the consequent slowing up of the defense program.

The physicians of Minnesota and of the nation are making an impressive contribution to defense preparations. They will do more, if more is asked of them; but when a welter of utopian schemes for improvement of civilian health is hastily tossed by civilian health officials into the already complicated machinery for registration, examination and induction into service of drafted men, then the physicians who will be asked to bear the burden are entitled to protest.

And when these schemes, if put into effect,

would also plunge the country into an endless program of government medical care of civilians, the obligation of physicians to oppose them is obvious.

The resolution passed by the Council at a special meeting held October 16 in Saint Paul is as follows:

1. WHEREAS, the Surgeon General of the United States Public Health Service has requested that serological tests for syphilis be given to every man who registers under the Selective Service Act, October 16, 1940, and

WHEREAS, the advisability of making the test on this scale has been thoroughly discussed with public health authorities and syphilologists in Minnesota:

IT IS HEREBY RESOLVED by the Council of the Minnesota State Medical Association called in special session on October 5, 1940, that the request to examine in this manner all registrants is impractical and inadvisable, especially in view of the emergency of the situation and the short time available to prepare for such an undertaking, and

WHEREAS, the registrants assigned to Class 1 when inducted into military service will have a complete examination at the mobilization center including a serological test:

A. It was the opinion of the Council that it would be inadvisable to duplicate such examinations for these registrants who are assigned to Class 1.

B. Since the selection and induction of men into service at this time is distinctly a military and not a public health program, it was the opinion of the Council that the procedures entailed by the test would lay an additional heavy and unnecessary burden and expense on physicians and public health services.

C. In view of the lack of organization for the collecting, mailing and testing of the specimens collected, it seemed advisable to avoid interjecting any procedure or undertaking that would complicate or in any way slow up the efficiency of the draft machinery.

2. WHEREAS, the program of the Minnesota State Medical Association and the Minnesota State Board of Health is a comprehensive one in that it includes not only syphilis but all infectious diseases, and

WHEREAS, encouragement of prophylactic and preventive medicine is part of that program,

It was the further opinion of the Council that such prophylactic measures should include prevention of venereal diseases at home and in the communities around the cantonment

3. WHEREAS, all deferred registrants under the selective Service Act still retain their civilian status.

It was also the opinion of the Council that any special testing or rehabilitation of those deferred registrants with regard to disease or disability should be carried out by the private physician with free choice of physician and hospital, thus helping to relieve overburdened military and public health facilities.

NATIONAL HEALTH PROGRAM—1940

That the National Defense program will also involve a national health program on a completely new basis seems altogether likely from the report of the September 16 Conference of State and Provincial Health Officers in Washington which was printed in *Public Health Reports* of the United States Public Health Service for September 27.

The customary cautiousness of United States Public Health Service officials in the handling of ticklish new plans for medical service to civilians was abandoned at this conference. A bright new world was envisioned. In this world, hands of the Health Service were to be freed, at last, to wipe out syphilis in the United States, to correct the physical defects of youth before they reached draft age and to rehabilitate the unfit discovered by draft board examiners—all without troublesome interference.

State medicine? Government medical service? Meaningless words now, it seemed; outmoded bogies. The mere invocation of those magic words, "National Defense" was to be enough, henceforth, to lay such uneasy wraiths.

They Took It For Granted

The surgeons of the United States Public Health Service put the problems to the conference and took for granted the answers. Of course, the program was desirable. Of course, the health service was ready to supervise, to supply funds to carry on the work. . . .

Presumably the conference acquiesced, at least informally, in the proposals made to it, though the method of securing acquiescence may have been similar to the method understood to have been used to gain backing for the astonishing proposal to offer every man who registered on October 16 a free Wassermann test for syphilis. This last-named plan was proposed to the conference by Senior Surgeon O. C. Wenger, of the Health Service.

Committee No. 2 Approved

In this case, it is understood, the regular conference committee of veneral disease was consulted first. This committee refused, because of the obvious difficulties involved, to give its approval; but its action was not al-

lowed to dampen the general enthusiasm. Another committee was promptly appointed and this second, impromptu committee approved without question. It was the report of the substitute committee that was presented to the conference.

"Discussion (of this plan) brought out a number of practical points and administrative difficulties," the health service account acknowledges, "such as the magnitude of the task to be accomplished within a very short time; the inadequate number of technicians available to perform the tests; the insufficient stock of specimen tubes and the lack of proper storage facilities for specimens awaiting examination."

According to the report, the health officers seemed anxious to accomplish "all that might be done within the limits imposed by circumstances."

Rejected for Minnesota

Actually, a compromise proposal was made by which Class 1 registrants, only, might be offered a Wassermann test. This proposal was later brought by Dr. A. J. Chesley, Secretary State Board of Health, to the official attention of the Council of the Minnesota State Medical Association for advice as to the policy which should be followed in Minnesota. The action taken by the Council at a special meeting, held, on both proposals and also on the proposal to provide rehabilitation for "culls," rejected from service, because of physical condition, is printed in full elsewhere in these columns. It will be noted that the Council did not favor even the compromise plan for several reasons, chief among them being the fact that selection and induction of men is distinctly a military and not a public health program, that testing Class 1 registrants would duplicate tests that would be made as a matter of routine at the time selectees were inducted into the service and that the procedure would place an added and unnecessary burden upon examining physicians.

The plan for providing physical examinations and correction of remediable defects among youths enrolled for benefits under the National Youth Administration was presented for approval to the Conference and has also been broached for Minnesota beneficiaries by director Chester A. Lund of Minnesota.

Youth Plans to Be Discussed

Just how the plan will eventually be conducted—if it is conducted in Minnesota—has not yet been determined. An early meeting with the Minnesota State Medical Association's Committee on Low Income and Indigent Problems, of which Dr. W. A. Coventry of Duluth is chairman, had been arranged as this issue went to press. It is altogether probable, however, that the Youth Administration will try to insist upon letting this work to the contract basis. State health departments are lowest-physician bidder in each county, on a contract basis. State health departments are to be asked to be co-sponsors in this work.

Following is the resolution introduced and, at least informally, approved by the conference on the subject of rehabilitating the men disqualified for military service because of physical defects.

To Correct Defects

RESOLVED: That persons who are otherwise found to be satisfactory and available for induction into the land or naval forces of the United States for training and service as provided by the Selective Training and Service Act of 1940, but who are placed on a deferred status because of physical defects or ailments which are readily amenable to treatment and cure may, upon application to the Surgeon General of the United States Public Health Service, be considered for acceptance as beneficiaries of that service for correction of such physical defects or ailments.

The committee contemplated, according to the report, that the Surgeon General of the United States Public Health Service would use his discretion in determining facilities, institutions and personnel to be employed in such corrective work and that any non-governmental facilities that might be used would be reimbursed out of funds made available to the United States Public Health Service for the operation of a program of rehabilitation among eligible rejected men.

Surgeons Thanked

Health of industrial workers, health and welfare of civilian populations, special public health measures involved in mobilization and in regions surrounding cantonments, education and qualifications of personnel—all these received attention and discussion.

At the end, the conference adopted a mild and non-commital formal resolution of thanks to the representatives of the United States Public Health Service, requested their con-

tinued interest and coöperation and drew attention of civil and military defense authorities to the importance of public health problems in the nation's defense.

If they did not formally go on record in favor of each resolution as it was presented, they certainly did not voice any objections—at least no objections were reported in the United States Public Health Service publication. The reason was, of course, that the object of the conference was not a new health program; instead it was national defense. And what social dreams if properly interpreted, cannot be tethered to national defense?

MEDICAL INDUCTION BOARDS

Profiting by experience of 1917, every man accepted for military service under the Selective Service Act of 1940 will be given a careful final physical examination by an Induction Board of specialists before he is assigned to cantonments or camps for training.

Tentative selections for appointments to Minnesota's board have already been made by Army Medical officers in consultation with medical association officials. All boards are to be made up of three internists, one general surgeon, one orthopedic surgeon, two opthalmologists, one otolaryngologist, one neuropsychiatrist, one clinical pathologist and one dentist, including probably both civilian specialists and reserve officers on temporary duty.

From nine to twelve of these boards will probably be needed for each corps area; but it is understood that only one will be needed for Minnesota and immediate vicinity. Headquarters for this board will be at Fort Snelling.

Service Temporary

Specialists selected will all be on temporary duty. They will probably serve for an estimated period of about a month following each selection of drafted men and they will probably be paid at the same rate of base pay as a major in the regular army, with allowances for travel and subsistence while on duty. Whether they will be required to be on duty for an entire eight-hour day or only for a part-time period each day had not yet been decided as this issue went to press.

"During mobilization," according to an editorial on the induction boards which appeared in a recent *Journal of the American Medical Association*, "the usual flow of men will be from local selective service boards to induction stations for final examination and induction, then to reception centers for classification and other processing, then to organization, installations or replacement centers in which they are to serve.

"The induction station serves the same purpose as the recruiting station does in times of voluntary enlistment. Therefore recruiting stations, augmented as required, will be used as induction stations."

Arrangements for x-ray, laboratory or other special examinations will be made as requested by the chief of the board in local hospitals or clinics.

Well Qualified Men Needed

It is obvious that thoroughly qualified men are needed for this board and that the service may involve a very real sacrifice on the part of members willing to offer themselves.

The importance to the effectiveness of our armed forces and also to the welfare of the individual drafted man, of painstaking examinations at this critical point in the progress from selection to training assignment goes without saying. It is made clearer when experience in the World War is taken into account. From the moment that the drafted man arrives at his training camp he becomes the responsibility of the government and, by the same token, any aggravation of ailments which he is permitted, by oversight, to take with him from civilian life is likely to become the basis for endless claims for compensation in time to come.

PRIVILEGED COMMUNICATIONS

(Monthly editorial prepared by the Medical Advisory Committee.)

Due to recent cases which have been brought to the attention of the Medical Advisory Committee, it is necessary that we again stress the fact that meticulous records, both office and hospital, made at the time of rendering service and signed by the physician or surgeon in charge himself constitutes one of the surest defenses against malpractice litigation; also that additions to these original records, if necessary, should under all circumstances be made, if possible, by the original writer, or at least, read and initialed by him. If not, the name of the writer of such additions should appear in full on the record.

These records should then be carefully preserved and made available at all times to the maker of them, but their contents should be con-

sidered as confidential information and at no time should they be exposed to public perusal unless the law or the courts demand it.

If privileged communications obtained in the physician-patient status are required by insurance companies, governmental agencies or any other party, they should only be divulged when the request is made in writing and such request signed by the patient or his legal representative, the request to be filed with the record together with copy of the report submitted.

Careful attention to this will become more and more necessary as government and state authorities, as well as private institutions, demand a greater quantity of information from the members of our profession.

The giving out of privileged communications and confidential information without proper authorization can become a distressing incident in the life of a physician.

—B.J.B.

THANKS OF THE COUNCIL

Special thanks and appreciation were expressed by the Council at their fall meeting to the following:

To Dr. J. M. Armstrong of Saint Paul, long chairman of the Historical Committee, for his faithful service and distinguished contributions to the collection, editing and publishing of manuscripts of the committee now appearing in MINNESOTA MEDICINE under the title "History of Medicine in Minnesota." Dr. Armstrong recently retired from the active practice of medicine in Saint Paul.

To the Olmsted - Houston - Fillmore - Dodge County Medical Society and to Mr. R. R. Rosell, executive secretary of the Minnesota State Medical Association, and his staff for their successful conduct of the 87th annual meeting of the Association held at the Mayo Civic Auditorium in Rochester last April. The final report of that meeting presented to the Council showed a substantial profit which has been turned over to the general fund of the Association.

PRICE OF HEALTH

The statement that the medical or health bill for society is too large is but an expression of the reluctance of society to pay the price for good health, as well as an evidence of complete failure to place full value on man's greatest asset—good health.

—FRANCIS F. BORZELL, M. D., in *Pittsburgh Medical Bulletin*, October 26, 1940.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

J. F. Du Bois, M.D., Secretary
Saint Paul, Minnesota

Itinerant Health Lecturer Pleads Guilty to Fraudulent Advertising and Practicing Healing Without a Basic Science Certificate

Re: State of Minnesota vs. Edward F. Marcell
Re: State of Minnesota vs. Guy Pearson

On October 5, 1940, Edward F. Marcell, fifty-three years of age, who gave Pasadena, California, as his home, entered a plea of guilty in the District Court of Morrison County, to an information charging him with the crime of practicing healing without a basic science certificate. The Honorable Don M. Cameron of Little Falls, Minnesota, Judge of the District Court, sentenced Marcell to pay a fine of $400.00 plus costs of $5.18, or to serve one year in the Morrison County Jail. Marcell paid the fine and costs. Marcell's brother-in-law, Guy Pearson, 31 years of age, of North Power, Oregon, pleaded guilty to assisting Marcell, and was sentenced by Judge Cameron, at the same time, to pay a fine of $50.00 and costs of $5.19, or to serve 90 days in the Morrison County Jail. Pearson paid the fine and costs.

Following a disposition of their cases in the District Court, Marcell and Pearson were arraigned before the Honorable Phil S. Randall, Judge of the Municipal Court of Little Falls, on a charge of fraudulent advertising. Both defendants pleaded guilty and each was sentenced by Judge Randall to pay a fine of $100.00 and costs of $3.10, or to serve ninety days in the Morrison County Jail. This sentence was suspended by Judge Randall because both defendants had paid their fines in the District Court, and upon the further condition that the defendants refrain from practicing healing in any manner in the State of Minnesota. The defendants are also to refrain from inserting any further advertisements in Minnesota papers, and in addition, they are to leave the State of Minnesota by October 15, 1940.

Marcell and Pearson arrived in the State of Minnesota about September 9, 1940, and proceeded to advertise a so-called exposé of devils, drugs and doctors at Detroit Lakes, Minnesota. They advertised a so-called health lecture for women only, following which a lecture was to be given for men only. The matter was called to the attention of the Minnesota State Board of Medical Examiners, but too late to apprehend the defendants at Detroit Lakes. Subsequently the defendants operated at Bemidji, Virginia and Little Falls, where they were arrested on October 4, 1940, following an investigation made by F. Manley Brist and Reginald M. Johnson on behalf of the Minnesota State Board of Medical Examiners, in coöperation with County Attorney Austin L. Grimes, Sheriff William J. Butcher and Deputy Sheriff Henry A. Smith. This investigation disclosed that the true purpose of inserting the advertisement was to secure prospective purchasers of a so-called health course which Marcell was selling at $58.00 per patient. Marcell recommended this health course as a treatment for a multitude of human ailments and claimed that it consisted of various mineral preparations in capsule form. As a special inducement, the health course could be purchased for $36.00 cash while Marcell was in Little Falls. The defendant's manner of operation, including the deceit practiced upon the public in the advertisement, and the numerous false statements made in the advertisement inserted in the Little Falls newspaper, stamp the defendants' activities as quackery. Both defendants readily admitted

that they had no medical training whatsoever. Marcell stated that he was in the commercial photography business for a number of years, and that in August 1939, his father died at Pasadena, California. He stated that his father had operated this so-called health course for many years.

The Minnesota State Board of Medical Examiners is very much pleased with the disposition made of these cases by Judge Cameron and Judge Randall. It, perhaps, is hardly necessary to point out that the imposition of a few stiff fines, such as were imposed in these cases, will go a long way toward stamping out this type of quackery. Minnesota has been comparatively free of this evil. Nevertheless, one or two other persons have operated in this State, and needless to say that other prosecutions are contemplated. The Minnesota State Board of Medical Examiners wishes to acknowledge the very prompt and efficient coöperation given in these cases by the local authorities, and particularly by County Attorney Grimes, Sheriff Butcher, and Deputy Sheriff Smith, all of Morrison County.

Minneapolis Woman Sentenced to Eight-Year Term at Hard Labor for Criminal Abortion

Re: State of Minnesota vs. Hattie Anderson.

On October 16, 1940, Mrs. Hattie Anderson, forty-five years of age, entered a plea of guilty to an information charging her with the crime of abortion. Due to the fact that the defendant had a previous conviction in 1933, for a similar offense, Mrs. Anderson was sentenced by the Honorable Paul S. Carroll, Judge of the District Court of Hennepin County, to a term of not to exceed eight years at hard labor in the Women's Reformatory at Shakopee.

Mrs. Anderson, who holds no license to practice any form of healing in the State of Minnesota, stated that she lived at Mound, Minnesota. On September 22, 1940, she performed a criminal abortion upon a twenty-one year old Wisconsin girl. The abortion was performed at Apartment 3, 89 Spruce Place, Minneapolis. Mrs. Anderson was paid $25.00 for her services. The girl became critically ill and was taken to Deaconess Hospital where she died on Tuesday, October 8, 1940. Because of the fact that the case was promptly reported by the attending physicians to the Minnesota State Board of Medical Examiners, it was possible, with the coöperation of the Women's Bureau of the Minneapolis Police Department, to obtain a statement from the girl before she died. The defendant was immediately placed under arrest and taken to the Hospital where she was identified by the girl just a few hours prior to her death.

Mrs. Anderson had a previous conviction for a similar offense on April 11, 1933, on which date she entered a plea of guilty in the District Court of Hennepin County to an information charging her with the crime of abortion. She was sentenced at that time, to a term of not to exceed eighteen months in the Women's Reformatory, but was placed on probation. She violated her probation by performing a subsequent abortion, and on February 21, 1936, her stay of sentence was vacated by Judge Baldwin. Mrs. Anderson served fourteen months and eighteen days for that crime. In the instant case it was decided to charge the defendant with the crime of abortion rather than manslaughter, because of her previous conviction, the laws of the State of Minnesota providing that where a defendant has one previous conviction, the Court may double the sentence for a subsequent offense. Mrs. Anderson attempted to have the Court limit her sentence to four years, but Judge Carroll told her that she had been shown considerable leniency by the State of Minnesota when she was charged with the crime of abortion rather than manslaughter.

Communication

MINNESOTA MEDICINE
Saint Paul, Minnesota
Gentlemen:

Men of medical and dental professions should be on guard against an imposter who goes about "borrowing" money. He represents himself variously as a physician, a dentist, or a son of a physician (depending on whom he is striking for a loan), as practicing in various small towns of the state and giving various names (Dr. Claud Mays, Rush City, Minn. when he interviewed me).

His story varies but in the above interview he was on his way home, had his wife and baby with him and would I lend him two dollars (his request is usually from two to five dollars) to get home on and he would send it back to me the next day. He disappeared while I was telephoning to check up on him.

He has a great deal of information about physicians indicating that he may have a medical directory to consult. He rushes in, greets with a glad hand, speaks very rapidly and glibly and has been very successful in victimizing many physicians and dentists in Duluth on at least three occasions in the past two years. A man answering his description has also operated on the Iron Range as well as in Superior, Wis. A man also answering his description has gone from house to house in Duluth selling "reducing pills" "recommended by the doctors at Rochester, Minnesota." He asks $1.50 down and the pills to be delivered in a week at which time the customer was to pay another $1.50 for the bottle.

This man is short, perhaps 5 feet 4 to 6 inches. He is very stocky and heavily built, has dark hair, and a very round face with plump cheeks. My impression is that he had a scar on the right side of the forehead. He was dressed in a wrinkled faded blue or blue-green suit.

By being forewarned, quick action on the part of the intended victim and coöperation with local authorities may result in the arrest and conviction of this imposter.

Yours truly,
L. L. MERRIMAN, M.D.
923 Medical Arts Bldg.
Duluth, Minnesota
October 3, 1940.

THE GANG THAT RUNS THINGS

"Ah, what's the use?" said the enthusiastic young physician, "I tried to horn in here, but only busted my horn! There is a gang that runs this medical society, and outsiders have no more show than a snake has hips!"

The Past President, to whom this remark was directed, lighted an obese cigar with a red and gold life preserver around its midriff and then spoke:

"You are right, young man, and you are wrong," he said. "By this statement I mean that you are right about a 'gang' running the society. There's a 'gang' running every organization on earth. If there wasn't there wouldn't be any organization. On the other hand, you are wrong when you say you tried to horn in and couldn't. You did not go about it right. Any good guy who wants to horn into any gang has to know the password, and I will give it to you in strictest confidence. Those words are: 'What can I do to help?' Any man who will use those words will find himself as busy as a one-armed piano player with a bad case of hives!"

—Detroit Medical News.

OF GENERAL INTEREST

At Ruthton, Minnesota, Dr. Arthur F. Sether is a member of the village council.

* * *

Dr. W. R. Morrison of Billings, Montana, recently visited in Bemidji, Minnesota, where he practiced for eight years, from 1900 to 1908, before going to Billings.

* * *

Dr. Paul F. Dwan of Minneapolis, who is recuperating after a severe illness, will leave about December 1 with his family for Florida to spend the winter.

* * *

Dr. Myron O. Henry of Minneapolis was guest speaker at the Camp Release Medical Society meeting at Dawson, Minnesota, October 11. His subject was "Treatment of Fractures of the Upper Extremity."

* * *

Dr. H. S. Diehl and Dr. C. D. Creevy of the Medical School, represented the University of Minnesota at the meeting of the Association of American Medical Colleges at Ann Arbor, Michigan, October 28-30.

* * *

Dr. Seigfried C. G. Oeljen of Waseca has moved into his newly completed office building north of the court house. The building, which houses only his offices, is of brick and is 22x47 feet.

* * *

Dr. F. G. Gunlaugson, health officer of the Mankato district office of the Minnesota Department of Health and a first lieutenant in the medical reserve officers corps, has been called to active duty for the next year. He is stationed at Fort Snelling.

* * *

Dr. George Michael Jennings, chief surgeon at the Northern Pacific Beneficial Association Hospital in Missoula, Montana, and a graduate of the University of Minnesota Medical School '08, passed away October 16.

* * *

Dr. A. E. Hansen, assistant professor of pediatrics at the University of Minnesota Medical School, addressed the annual meeting of the Bryan Memorial Hospital in Lincoln, Nebraska, October 8, on the subject of "Rheumatic Fever."

* * *

The marriage of Dr. J. J. Stratte of Grand Forks, North Dakota, to Miss Irene Dille, took place September 30. Dr. Stratte, former Kittson County surgeon, in addition to his practice in Grand Forks, is associated with the Berlin-Griffin Clinic at Hallock, Minnesota.

Dr. George C. Kimmel, Mayo Foundation fellow, will become associated with the Interstate Clinic at Red Wing, January 1, it is announced. Graduated from the University of Minnesota Medical School in 1936, Dr. Kimmel took his internship at Temple University in Philadelphia.

* * *

Dr. Bernard Watson, who left the staff of the University of Minnesota Health Service and Medical School last spring to become associated with the Battle Creek Sanitarium at Battle Creek, Michigan, visited in Minneapolis in October, while in the state on a pheasant hunting trip.

* * *

Dr. and Mrs. George B. Weiser of New Ulm marked their fiftieth wedding anniversary, October 2. They were married in 1890 at Selinsgrove, Pennsylvania. Their son-in-law and daughter, Mr. and Mrs. C. T. Ekelund of Pontiac, Michigan, came to New Ulm for the celebration, at which the couple received the congratulations of their many friends.

* * *

Dr. Russell W. Morse of Minneapolis was installed as president of the Hennepin County Medical Society, October 8. He succeeds Dr. James A. Johnson. Plans for the year include sponsorship of a series of lectures for expectant mothers in connection with the adult education program of the YWCA. Lectures will be given each Friday morning.

* * *

Dr. A. C. Ivy of Chicago will give the annual E. Starr Judd lecture at the University of Minnesota Medical Amphitheatre, January 15, 1941, at 8:15 p. m. His subject is "The Mechanisms of Gastric Secretion."

Dr. Ivy is head of the Department of Physiology and Pharmacology at Northwestern University Medical School.

* * *

Dr. Hans E. Guloien of Fargo, North Dakota, has located in Willmar, Minnesota, opening offices there at 316 West Fifth Street.

A graduate of Rush Medical School, University of Chicago, in the Class of 1938, Dr. Guloien served his interneship at St. Mary's Hospital in Duluth. Prior to coming to Willmar, he was resident surgeon at the Mary McClellan Hospital at Cambridge, New York.

* * *

A father and his son have become associated in the practice of medicine in Red Wing, Minnesota. Dr. L. E. Claydon, one of the oldest practicing physicians in

Goodhue county, and Dr. Donald R. Claydon have opened new offices at 324 Main Street. Also associated with them is Dr. E. M. Baldigo, a graduate of the University of Minnesota Medical School, who served his internship at Miller hospital in Saint Paul.

* * *

Dr. and Mrs. William M. Balfour of Rochester, New York, are the parents of a son, James McQuarrie Balfour, born August 26. Dr. Balfour, who is a research fellow in pathology at the University of Rochester Medical School, is the son of Dr. and Mrs. Donald C. Balfour of Rochester. Mrs. Balfour, the former Oane McQuarrie, is the daughter of Dr. and Mrs. Irvine McQuarrie of Minneapolis.

* * *

Dr. R. F. Pierson, licensed in Minnesota by reciprocity with Nebraska, has become associated with Dr. Henry C. Doms of Slayton, Minnesota. Dr. Pierson, who was graduated from the University of Nebraska in June, 1938, served one year as an intern at the Clarkson Hospital in Omaha, and six months at the Fairview Hospital in Minneapolis.

Dr. Pierson married Miss Maxine Steggs, a graduate of the Clarkson Hospital School of Nursing, April 7, 1940.

* * *

The University of Minnesota Medical School has instituted a required course in First-Aid for freshmen medical students. The one-quarter course, consisting of fourteen lectures and six practical demonstrations, was instituted because it was felt that it was pertinent to the preparedness program in event of an emergency. The University of Minnesota is one of the first medical schools in the country to include such a course for freshmen in the curriculum. Ordinarily, medical students do not receive any instructions in caring for emergencies until they are juniors and seniors.

* * *

Dr. Bertram S. Adams, president of the Minnesota State Medical Association, was honored by the Hibbing Elks Lodge and citizens of Hibbing for his civic service to the community and the state, October 22. The testimonial dinner, an outstanding event in northeastern Minnesota, was the Elks' seventh annual honor night.

More than 400 men, many of them distinguished leaders of the medical and dental professions and of fraternal societies, attended the dinner. In addition to verbal tributes, there were scores of congratulatory telegrams and communications, among them one from Governor Harold E. Stassen and another from the employees of the Adams Hospital.

* * *

Dr. A. B. Stewart of Owatonna, "dean" of the medical and surgical profession of Steele County, was the recipient of many congratulatory messages when he observed his seventy-seventh birth anniversary, September 23.

Born at Hume, New York, in 1863, Dr. Stewart has practiced in Owatonna since June, 1891. He has studied at hospitals and clinics in Vienna, and Edinburgh, and holds a fellowship in the American College of Surgeons. In 1918 he was given the rank of major in the U. S. Army and was chief of the medical staff at Fort Snelling.

* * *

Dr. B. T. Horton of Rochester addressed a meeting of the Tennessee Valley Postgraduate Medical Assembly in Knoxville, Tennessee, October 10. He presented two papers: "The Treatment of Peripheral Vascular Disease" and "Histamine in the Treatment of Specific Types of Headache." On October 14, he addressed a meeting of the Norfolk County Medical Society in Norfolk, Virginia, speaking on "The Treatment of Headaches and Vertigo with Histamine." From there he went to the University of Virginia in Charlottesville to speak before a meeting of the Virginia Neuropsychiatric Society.

* * *

When the fifteenth annual conference of the Association of Clinic Managers is conducted in Rochester, November 6, 7 and 8, the program will include an address of welcome by Dr. C. W. Mayo; an address, "The Value of Medical Statistical Research" by Dr. Joseph Berkson of Rochester; a talk on "Medico-military Preparedness" by Dr. Russell M. Wilder of Rochester, chairman of the Committee on Medicine in the Division of Medical Sciences of the National Research Council; and an address, "Air Conditioning: Its Use and Limitations in Clinics and Hospitals" by Dr. Charles Sheard of Rochester.

* * *

Three experiments, designed to engender and conserve energies and resourcefulness of the United States Army, are being carried on in the University of Minnesota physiological laboratories under the direction of Dr. Ancel Keys, professor of physiology. One of the projects has to do with the study of fatigue in soldiers in marching and field operations, and with means of reducing the fatigue and increasing ability to carry on maneuvers.

A second experiment concerns the prevention and treatment of wound shock, especially in emergencies arising on the battlefield; the third, with a study of heart and circulation under conditions of low oxygen supply, as found in planes flying at high altitudes.

* * *

Two Minnesota physicians were among the prize winners in the Abbott Camera Competition for Physicians, and two others received honorable mention.

Dr. Lawrence Berman of Saint Paul was one of the

fourth prize winners in the pictorial and general division with his entry, "Hilltop." Dr. Charles Heilman of Rochester and Dr. Ernest J. Losli of Minneapolis were among those who received honorable mention. Dr. A. J. Hertzog of Eau Claire, formerly of the University of Minnesota Medical School staff, won an honorable mention.

Dr. J. R. Sturre of Minneapolis won third prize in the division devoted to Gross Specimens and Photomicrographs.

* * *

Dr. Irvine McQuarrie, head of the Department of Pediatrics at the University of Minnesota Medical School, has returned from Rochester, New York, where he addressed two meetings. He spoke at the Rochester Academy of Medicine, October 15, his subject being "Medical Conditions in North China As Related to Military Invasion." On October 16 he addressed the Rochester Pediatrics Society on "Adrenal Tumors in Childhood."

Dr. McQuarrie on September 18 and 19 gave two lectures at the University of Iowa in Iowa City for a program on Maternal and Child Hygiene, sponsored by the State Board of Health. His subjects were "Anoxia and Asphyxia in the New Born" and "Common Diseases of the New Born Infant."

* * *

Physicians who are lecturing in the course, "Orientation to Practice," offered to seniors in the University of Minnesota Medical School, include: Dr. H. S. Diehl, "Opportunities In and Preparation for Practice"; Dr. A. W. Adson of Rochester, "Medical Licensure"; Dr. W. A. O'Brien, "Quackery, Fads, Cults and Patent Medicines"; Dr. F. J. Hirschboeck of Duluth, "The Ethics of the Practice of Medicine"; Dr. S. M. White, "The Management of the Public and Private Patient"; Dr. J. M. Hayes of Minneapolis, "Starting the Practice of Medicine"; Dr. B. J. Branton of Willmar, "Malpractice"; Dr. R. E. Scammon, "Medical Care of the Indigent and of Low Income Groups." Judge Paul Carroll of Minneapolis is lecturing on "The Physician in Court."

* * *

Representing the University of Minnesota at the American Public Health Association meeting in Detroit, Michigan, in October were: Dr. Ruth E. Boynton, director of the Students' Health Service; Dr. Ralph V. Ellis, associate professor of preventive medicine; Dr. Gaylord Anderson, head of the Department of Preventive Medicine and Public Health; Miss Mellie Palmer, acting director of the course in Public Health Nursing; George O. Pier, assistant professor; and Miss Mary Parker.

Though he was unable to attend, Dr. W. A. O'Brien was honored at the banquet, October 10. He was among the former members of the Detroit Department of Health, who were awarded certificates of merit for the outstanding work they have done elsewhere. The awards were made by Dr. Henry F. Vaughn, director of the Department of Health of the city of Detroit.

The first annual Minnesota Medical Foundation Lecture will be presented at the University of Minnesota Medical School on Tuesday evening, November 12. Dr. Conrad A. Elvehjem, Professor and Head of the Department of Biochemistry, University of Wisconsin, will present the lecture, the title of which is "The Biochemistry of the Vitamin B Complex."

* * *

The University of Minnesota Board of Regents has accepted a gift of a collection of medical journals from Dr. Ivar Siversten of Minneapolis.

* * *

Dr. Morris Fishbein was principal speaker at the General Medical Faculty annual dinner meeting in the new Coffman Memorial Union on the University of Minnesota campus, October 4. He spoke on "Medical Writing" and "The Role of the Medical Profession in National Defense."

Dr. H. S. Diehl presided at the dinner, the program for which also included talks by Dr. James S. McCartney on the admission of students to the Medical School during the current year; a report by Dr. Erling S. Platou, president, on the development of the Minnesota Medical Foundation during its first year; a report by Dr. W. A. O'Brien on postgraduate medical courses during the past year; observations by Dr. Irvine McQuarrie on his recent visit in China, when he held a visiting professorship at the Peiping Union Medical College. Dr. Diehl reported on the organization of the United States General Hospital Unit No. 26.

* * *

The 1941 convention of the Interstate Post Graduate Medical Association of North America to be held in Minneapolis next October, is expected to bring 4,000 physicians and surgeons to that city.

The association recently concluded its 1940 assembly in Cleveland, Ohio. Speakers at the meeting, held from October 14 through 18, included several Minnesotans: Dr. Maurice B. Visscher, head of the physiology department at the University of Minnesota, whose subject was "Intestinal Absorption as a Clinical Physiological Problem"; Dr. Walter C. Alvarez of Rochester, who conducted a diagnostic clinic, "Puzzling Cases of Abdominal Pain"; Dr. Waltman Walters of Rochester, diagnostic clinic, "Surgery of the Pancreas"; Dr. Howard K. Gray of Rochester, address, "Cancer of the Stomach"; Dr. Charles W. Mayo of Rochester, address, "The Treatment of Vesico-Colonic Fistula"; Dr. Claude F. Dixon of Rochester, diagnostic clinic, "Diverticulitis"; Dr. Alfred W. Adson of Rochester, diagnostic clinic, "Surgical Treatment of Peripheral Vascular Disease"; Dr. William F. Braasch of Rochester, address, "Infections of the Urinary Tract"; Dr. John S. Lundy of Rochester, address, "Choice of Anesthesia."

Dr. William L. Benedict of Rochester gave the Schneider Foundation Eye Presentation. The subject of his address was "Relationship of Ophthalmology to Systemic Disease."

The first permanent board of trustees for the Minnesota Medical Foundation was elected at the organization's first annual meeting, October 25, in the new Coffman Memorial Union on the University campus.

Elected to the board of twelve members to serve terms of four years each were Dr. William W. Will of Bertha, Dr. Edwin J. Simons of Swanville, Dr. Lloyd H. Rutledge of Detroit Lakes; to serve three-year terms, Dr. Russell J. Moe of Duluth, Dr. Albert M. Snell of Rochester and Dr. Gordon C. MacRae of Duluth; two-year terms, Dr. Jennings C. Litzenberg, Dr. Owen H. Wangensteen and Dr. Cecil J. Watson of Minneapolis; and one-year terms, Dr. Erling S. Platou of Minneapolis, Dr. George A. Earl of St. Paul and Dr. Robert L. Wilder of Minneapolis.

Dr. Platou, president of the Foundation, presided at the meeting and reported on the program and development of the Foundation during the past year. Dr. Wilder gave the secretary's report, and Dr. Litzenberger, the treasurer's report. Reporting on membership expansion activities were Dr. Will, Dr. Moe and Dr. Olga Hansen. Dr. Maurice B. Visscher, editor of *The Bulletin of the Minnesota Medical Foundation*, reported on that publication.

Dr. Erling W. Hansen reported for the Nominating Committee.

* * *

Speakers at the twelfth annual meeting of the Medico-Military Inactive Status Training Unit in Rochester October 6-20, included besides members of the Mayo Clinic staff: Colonel Kent Nelson, MC USA, Medical Director R.O.T.C. University of Minnesota; Major F. B. Waweman, MC USA, Washington, D. C.; Lt. Comdr. Donald McCarthy, MC-O, USNR, Minneapolis; Lt. Albert R. Behnke, Jr., MC USN, Washington; Captain Andrew H. Davidson, MC USN, District Medical Officers 9th Naval District, Great Lakes, Illinois.

Also Lt. Col. Jarret M. Huddleston, MC USA, Fort Snelling, Minnesota; Colonel Monte Belot, Med. Res., Kansas City, Missouri; Comdr. John R. Poppen, MC USN, Bureau of Aeronautics, Navy Department, Washington, D. C.; D. R. Brimhall, Director of Research, C.A.A., Washington; Dr. H. S. Diehl of the University of Minnesota; Brig. Gen. John H. Hester, Inf. Executive for Reserve Affairs, War Department, Washington.

Also Colonel A. D. Tuttle of United Airways, Chicago; Lt. Herbert M. Bosch of the Minnesota State Board of Health, Minneapolis; Major James J. Morrow, Med. Res. of Austin; Lt. Comdr. R. H. Hunt, MC USN, Minneapolis; and Dr. Edward A. Dosey, Professor of Bio-Chemistry and Director of the Department, St. Louis University Medical School, St. Louis, Missouri.

The staff consisted of Dr. Donald C. Balfour, director of the Mayo Foundation; Col. Herbert C. Gibner, MC USA, Corps Surgeon, 7th Corps Area, Omaha; Captain Ernest W. Brown, MC USN, Senior Medical Officer, Navy Yard, New York; Col. Frederick L. Smith, Med. Res. USA, Rochester; and Captain Robert O. Pearman, Med. Res., St. Joseph, Missouri.

In Memoriam

John Rishmiller

Dr. John Rishmiller, Minneapolis surgeon for many years, passed away at the home of a nephew in Gibbon, October 15. He was seventy-four years old.

Dr. Rishmiller was chief surgeon of the Soo Line Railway in Minneapolis for a long period, but severed his connection several years ago when he retired from practice. He had made his home in Gibbon since last spring.

Dr. Rishmiller was vice president of the Hennepin County Medical Society in 1912.

Charles Alex Van Slyke

Dr. Charles Alex Van Slyke, one of the first ophthalmologists to practice in Saint Paul, died Sunday, July 21, 1940.

Dr. Van Slyke's father, William A. Van Slyke, came to Minnesota Territory in 1859, was active in politics, and is said to have been the father of the park system in Saint Paul.

Charles A. Van Slyke was born December 14, 1865. He received his education at the Franklin School and later attended Shattuck Military Academy at Faribault where he was a member of the football and baseball teams. In 1887 and 1888 he attended medical lectures at the Saint Paul Medical College and in 1889 at Cooper Medical College, San Francisco.

In the winter of 1890, Dr. Van Slyke made a trip to the South Sea Islands. He wrote a short description of the trip and made an interesting collection of shells from the islands.

Dr. Van Slyke received his medical diploma from the Medical School of the University of Minnesota in 1891. He spent part of the year 1892 in study abroad. Upon his return he studied during the years 1893 to 1895 at the New York Ophthalmic and Aural Institute. He was house officer at Knapp Hospital for Eye, Ear, Nose and Throat in 1894. He then returned to Saint Paul and began the practice of his specialty with his brother, Dr. Fred Van Slyke. He had offices in the Kendrick Building, Baltimore Block, Endicott Building and the Lowry Medical Arts Building. He was in active practice until 1936 when he sustained a cerebral accident. He later established an office at his residence. A second cerebral accident in 1940, followed by a hip fracture from a fall, proved fatal.

Dr. Van Slyke belonged to the Saint Paul Rod and Gun Club for many years and was president of that club in 1911. He was a member of the Royal Arch Masons for many years and was a thirty-second degree Mason and a Knight Templar. He was president of the Junior Pioneers in 1919. In early life he was a member of the Minnesota National Guard.

Dr. Van Slyke was a member of the Minnesota Academy of Ophthalmology, the Ramsey County Medical Society, the Minnesota State and American Medical Associations.

Dr. Van Slyke married Anna C. Perrior in Saint Paul, December 15, 1902. He is survived by his widow, a son, Charles Alex Van Slyke, Jr., of Milwaukee and a daughter Mrs. Ralph Coggeshall of Plainfield, New Jersey, a brother, Kenneth, of Saint Paul and a sister, Grace, of Saint James, Minnesota.

He had many loyal patients and friends.

—JAMES C. FERGUSON.

J. Thornwell Witherspoon

Dr. J. Thornwell Witherspoon, a member of the Hennepin County Medical Society and editor of *Modern Medicine*, passed away recently at the Norwalk Hospital in Norwalk, Connecticut.

He was a contributor to medical literature, having published more than fifty articles and many abstracts in leading gynecological and surgical journals. His latest medical textbook, "Clinical Pathological Gynecology," was published in 1939.

Born in New Orleans in 1900, he was graduated from Princeton University and spent two years at Oxford on a Rhodes scholarship. He received his M.D. degree at Johns Hopkins University.

Victor A. Young

Dr. Victor A. Young passed away August 20, 1940 at his home in West Duluth, Minnesota, after an illness of three years.

He was a native Minnesotan, born at Amor, Minnesota, April 21, 1875. In 1903 he received his M.D. degree at Hamline University. The year before his graduation he was married to Mary Alice Jones, a daughter of a pioneer physician, at Battle Lake, Minnesota. This companionship was interrupted by the death of Mrs. Young in February, 1930. The responsibility of two daughters and two sons fell on Dr. Young's shoulders. In August, 1935, he married Susan A. Isaacson of Fergus Falls, Minnesota, who together with his children survive him.

Dr. Young's first practice was at Scanlon, Minnesota, which, at that time, was a lively lumbering town. Here he remained for two years and then moved to Hankinson, North Dakota. In 1915 he located in West Duluth, Minnesota, and continued his practice there until his death.

Dr. Young will be remembered for his devotion to his work and his kind sympathy and understanding.

REPORTS and ANNOUNCEMENTS

MEDICAL BROADCAST FOR NOVEMBER

The Minnesota State Medical Association broadcasts weekly at 11:00 o'clock every Saturday morning over Station WCCO, Minneapolis, Station WLB, University of Minnesota, and KDAL, Duluth.

Speaker: William A. O'Brien, M.D., Professor of Preventive Medicine and Public Health, Medical School, University of Minnesota.

The program for the month will be as follows:

November 2—Eye Injuries
November 9—Glaucoma
November 16—The Crosseyed Child
November 23—Cataracts
November 30—Children's Dentistry

UNIVERSITY OF CHICAGO POSTGRADUATE COURSES

The Department of Obstetrics and Gynecology of the University of Chicago and the Chicago Lying-in Hospital, through the coöperation of the Children's Bureau, U. S. Department of Labor and the Illinois State Department of Public Health, offers five postgraduate courses of four weeks each between January 6 and June 21. The beginning dates of each are: January 6, February 10, March 17, April 21, and May 26. All the members of the department and all services and units of the institution participate in the instruction. Only a limited number of postgraduate students are accepted for each period. A deposit of $25.00 is required, of which $10.00 is returned on completion of the course. All communications should be addressed to: Postgraduate Course, 5848 Drexel Avenue, Chicago, Illinois.

AMERICAN PUBLIC HEALTH ASSOCIATION

The registered attendance of the 69th Annual Meeting of the American Public Health Association and meetings of related organizations held in Detroit the week of October 7 was 3,187, second largest in the Association's history. Delegates came from every State in the Union, the District of Columbia, Alaska, Hawaii, Puerto Rico, Canada, Cuba, Mexico, Denmark, China and New Zealand.

The officers elected for the year 1940-1941 are as follows:

President: W. S. Leathers, M.D., Nashville, Tenn.
President-elect: John L. Rice, M.D., New York, N.Y.
Vice President: Robert D. Defries, M.D., Toronto, Can.
Vice President: Charles Edward Finlay, M.D., Havana, Cuba.
Vice President: Selskar Gunn, New York, N. Y.
Treasurer: Louis I. Dublin, Ph.D., New York, N. Y.

Chairman of Executive Board: Abel Wolman, Dr. Eng., Baltimore, Md.

Executive Secretary: Reginald M. Atwater, M.D., New York, N. Y.

A Committee on Public Health in the National Defense was appointed with the following personnel: W. S. Leathers, M.D., Chairman; Stanley H. Osborn, M.D.; Huntington Williams, M.D.; Abel Wolman, Dr. Eng.

Among the resolutions passed was one emphasizing the necessity for maintaining civilian health as essential in national defense and pledging the united support of members to the national defense and to the maintenance of health in a free people.

The 70th Annual Meeting will be held in Atlantic City, N. J., in October, 1941.

ALUMNI ASSOCIATION OF THE MAYO FOUNDATION

Dr. Porter P. Vinson of Richmond, Virginia, was elected president of the Alumni Association of the Mayo Foundation at its twenty-second annual meeting in Rochester last month.

Other officers are Dr. William H. Long of Fargo, North Dakota, first vice president; Dr. J. Blackford of Seattle, Washington, second vice president; Dr. Ernest L. Meland of Minneapolis, secretary. Dr. D. M. Masson of Rochester was reëlected associate secretary and treasurer. Dr. John M. Berkman of Rochester was elected to the board of governors.

Named to the advisory board for three-year terms were Dr. J. M. Culligan of St. Paul, Dr. George D. Mahon of Dallas, Texas, Dr. Robert W. McQuay of Toronto, Dr. Charles H. Leech of Lima, Ohio, and Dr. Lindon Seed of Chicago.

MINNESOTA PATHOLOGICAL SOCIETY

Two papers will be presented at the meeting of the Minnesota Pathological Society, November 19, at 8 p. m. in the amphitheater of the Institute of Anatomy at the University of Minnesota. Dr. R. G. Green will present a paper, "The Nature of Virus Infections," and Dr. John M. Adams, "Epidemic Virus Pneumonia in New-Born Infants."

WABASHA COUNTY MEDICAL SOCIETY

Dr. D. G. Mahle of Plainview was elected president of the Wabasha County Medical Society, succeeding Dr. E. W. Ellis of Elgin, at the society's annual meeting in Plainview, October 10.

Dr. Robert A. Glabe of Plainview was elected vice president, and Dr. W. F. Wilson of Lake City was re-elected secretary-treasurer. Dr. E. C. Bayley of Lake City was named a delegate to the State Association, and Dr. Ellis of Elgin was named alternate. The board of censors is composed of Dr. R. R. Hendrickson of Wabasha, chairman; Dr. Mahle, and Dr. W. J. Cochrane of Lake City.

The business session preceded a duck dinner, at which there were thirty-three guests. Following the dinner, there was a scientific session.

WEST CENTRAL MEDICAL SOCIETY

At the annual meeting of the West Central Medical Society held in Morris, October 9, Dr. F. W. Behmler of Morris was elected president, and Dr. Otto Bergan of Clinton, vice president. Dr. H. Linde of Cyrus was re-elected secretary-treasurer. Named as delegate to the State Association was Dr. C. I. Oliver of Graceville. Alternate is Dr. Charles Bolsta of Ortonville. Dr. N. F. Doleman of Tintah was elected censor for three years.

Dr. E. M. Elsey of Glenwood is the retiring president.

At the meeting a medical fee schedule was adopted by the society.

Dr. B. W. Jarvis of Lowry was accepted as a member of the society.

WOMEN'S AUXILIARY

Mrs. A. C. Baker, Fergus Falls, *President*
Mrs. E. V. Goltz, 2259 Summit Avenue, Saint Paul, *Publicity Chairman*

The first fall board meeting of the Women's Auxiliary of the Minnesota State Medical Association was held Friday morning, October 4, in the Center for Continuation Study at the University of Minnesota. Mrs. M. A. Nicholson, state president, presided. Following the business meeting, luncheon was served. Mrs. Wm. B. Roberts of Minneapolis was the social chairman for the day.

It was gratifying to have a splendid attendance. Considerable enthusiasm was shown and an eagerness for the new season's work. Members of the board were guests of the Hennepin County Auxiliary at the open meeting and tea held at the home of Mrs. J. F. Curtin on West Lake Boulevard, following the board meeting.

On Monday, September 30, Mrs. J. J. Ryan of Saint Paul was hostess at an informal breakfast in honor of the national president, Mrs. V. E. Holcombe, who was passing through Saint Paul.

Sincerest sympathy is extended to Dr. Richard R. Cranmer of Minneapolis in the recent death of Mrs. Cranmer who had endeared herself to members of the Auxiliary. Her untimely death, occurring after several weeks of illness, has been a blow not only to her own Hennepin Auxiliary, but to the state board as well. She was a most enthusiastic Auxiliary worker and was also active in the P.E.O. and other organizations.

Mower County Auxiliary recently held its first meeting which was followed by luncheon with twelve members present.

Washington County Auxiliary reports the election of new officers as follows: Mrs. F. M. McCarten, president; Mrs. Gertrude Stevens of Lake Elmo, vice president, and Mrs. C. H. Sherman of Bayport, secretary and treasurer. There were eleven members present at the meeting which was held at the home of Mrs. Landeen.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

OBSTETRICS IN GENERAL PRACTICE. J. P. Greenhill, B.S., M.D., F.A.C.S. Professor of Obstetrics and Gynecology, Loyola University Medical School, Chicago; Professor of Gynecology, Cook County Graduate School of Medicine; Attending Gynecologist, Cook County Hospital; co-editor Year Book of Obstetrics and Gynecology; author of Office Gynecology. 448 pages. Illus. Price, $3.50, cloth. Chicago: Year Book Publishers, 1940.

VITAMIN THERAPY IN GENERAL PRACTICE. Edgar S. Gordon, M.D., M.A. Associate in Medicine and Instructor in Physiological Chemistry, University of Wisconsin; and Elmer L. Sevringhaus, M.D., F. A. C.P. Professor of Medicine, University of Wisconsin, editor Department of Endocrinology, Year Book of Neurology, Psychiatry and Endocrinology. 258 pages. Illus. Price, $2.75, cloth. Chicago: Year Book Publishers, 1940.

THE 1940 YEAR BOOK OF PUBLIC HEALTH. Edited by J. C. Geiger, M.D., Dr.P.H. Director of Public Health, City and County of San Francisco; Clinical Professor of Epidemiology, University of California. 560 pages. Illus. Price, $3.00, cloth. Chicago: Year Book Publishers, 1940.

METHODS FOR DIAGNOSTIC BACTERIOLOGY. A Complete Guide for the Isolation and Identification of Pathogenic Bacteria for Medical Bacteriology Laboratories. Isabelle G. Schaub, A.B., Assistant in Bacteriology, Department of Pathology and Bacteriology, Johns Hopkins University School of Medicine; and M. Kathleen Foley, A.B., Bacteriologist in Charge of the Diagnostic Bacteriological Laboratory of the Medical Clinic, the Johns Hopkins Hospital, Baltimore. 313 pages. Priced, $3.00, cloth. St. Louis: C. V. Mosby Co., 1940.

THE PRACTICE OF MEDICINE. Third Edition. Jonathan Campbell Meakins, M.D., LL.D. Professor of Medicine and Director Department of Medicine, McGill University; Physician-in-Chief, Royal Victoria Hospital, Montreal; Formerly Professor of Therapeutics and Clinical Medicine, University of Edinburgh, etc. 1,430 pages. Illus. Price, $10.00, cloth. St. Louis: C. V. Mosby Co., 1940.

BACILLARY AND RICKETTSIAL INFECTIONS—Acute and Chronic. William H. Holmes. Professor of Medicine, Northwestern University Medical School; Chairman Department of Medicine, Passavant Memorial Hospital, Chicago. 676 pages. Price, $6.00, cloth. New York: The MacMillan Co., 1940.

SYNOPSIS OF MATERIA MEDICA, TOXICOLOGY AND PHARMACOLOGY. For Students and Practitioners of Medicine. Forrest Ramon Davison, B.A., M.Sc., Ph.D., M.B. Assistant Professor of Pharmacology in the School of Medicine, University of Arkansas. 633 pages. Illus. Price, $5.00, flexible binding. St. Louis: C. V. Mosby Co., 1940.

A TREATISE ON MEDICOLEGAL OPHTHALMOLOGY. By Albert C. Snell, M.D. Lecturer in Ophthalmology, School of Medicine and Dentistry, University of Rochester; Consultant in Ophthalmology, Strong Memorial Hospital and Rochester General Hospital; Ophthalmologist Park Avenue Hospital, Rochester, N. Y., etc. 312 pages. Illus. Price, cloth, $6.00. St. Louis: C. V. Mosby Co., 1940.

A TEXTBOOK OF MEDICINE BY AMERICAN AUTHORS. Edited by Russell L. Cecil, Professor of Clinical Medicine, Cornell University Medical College. Associate Editor for Diseases of the Nervous System, Foster Kennedy, Professor of Clinical Neurology, Cornell University Medical College. 5th Edition. Philadelphia and London: W. B. Saunders Co. 1940. Price $9.50.

Previous editions of the textbook have rightly earned great popularity, especially among younger members of the profession. The fifth edition, which has just appeared, contains a number of new articles on subjects not covered in previous editions and a number of rewritten articles on subjects previously covered. Other articles have been brought up-to-date and new illustrations have been added.

On the proper assumption that no one individual can know or present in a textbook all that is known in the field of medicine. the editor has obtained the services of some 130 teachers of medicine in university medical colleges who have written on various subjects of particular interest to them, and the result is a particularly fine volume for the undergraduate medical student or the medical practitioner. This method of textbook writing obviously adds to the up-to-dateness of a volume and that applies to the present volume. The editors are to be congratulated.

C. B. D.

CLINICAL HEART DISEASE. Samuel A. Levine, M.D., F.A.C.P., Assistant Professor of Medicine, Harvard Medical School. Second Edition. 495 pages. Illus. Price $6.00. W. B. Saunders Company, 1940.

Levine's second edition of his "Clinical Heart Disease" is a book written by a clinician for clinicians. It is a clear, concise monograph on modern cardiology. This is no treatise on cardiac physiology, nor does it contain unnecessary pages of references. It is a book which reflects years of experience in cardiology, and it gives to the reader the practical results of the author's knowledge. The book is well written and easy to read. Levine's monograph is highly recommended both for students and practicing physicians.

M. J. SHAPIRO, M.D.

SYMPTOMS OF VISCERAL DISEASE. F. M. Pottenger, A.M., M.D., LL.D., F.A.C.P. 5th edition. 442 pages. Illus. Price $5.00. St. Louis: C. V. Mosby Co., 1938.

The author is one of the real pioneers in a fundamental and consistently neglected field. In some teaching institutions his approach to the character of disease is almost heresy. His broad view axiomatically precedes the opening chapter in these words: "There is a patient who has the disease as well as the disease which has the patient."

Dr. Pottenger is a chronic abstainer from the evils of dogma and the empiric modus operandi. He is a sincere champion of the constructive evolution of medical practice. This is evidenced by one sentence in his

introduction to this edition: "Nearly every chapter has been partially or wholly rewritten." Those of us, whose privilege it is to know him, understand this to convey his consistent dissatisfaction with our limitations. He remains dissatisfied with such routine practice as considering a case complete by virtue of an accepted diagnostic name and, arbitrarily given, more or less effective accepted therapy. He cares more for the physiologic bedrock than the nominally polished but meaningless superstructure.

For simple illustration, some are appeased by such a term as "nervous stomach." He would like to know where the nervousness originates and how it operates. Is it central, sympathetic, parasympathetic, endocrine, humeral, or what, and if so, why?

Through all the areas and systems of the body this insatiable curiosity stalks. His answers are by no means complete or wholly adequate (nor are anyone's), but his search could be a source of stimulation to anyone with a will or a desire to pursue truth or allow a well deserved vacation to his suppressed analytic intellect.

ROBERT LYMAN NELSON, M.D.

OPERATIVE SURGERY. J. Shelton Horsley, M.D., LL.D., F.A.C.S., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va., and Isaac A. Bigger, M.D., Professor of Surgery, Medical College of Virginia, Surgeon-in-chief, Medical College of Virginia Hospitals, Richmond, Va. Fifth Edition. 2 Volumes. 1,567 pages. Illustrated by Helen Lorraine. Price $18.00. St. Louis: C. V. Mosby Company, 1940.

A two-volume work which covers the operative procedures of the authors and collaborators is not an attempt to include all surgical operations, but is rather to a considerable extent a record of the author's personal experiences.

Among the new procedures described are ligation of the patent ductus arteriosus, segmental pneumonectomy, and extrapleural pneumothorax. Segmental pneumonectomy is a recent development in the surgical treatment of bronchiectasis and extrapleural pneumothorax appears to have a distinct place in the surgical treatment of pulmonary tuberculosis. The chapter on peritonitis in which the pathology, symptoms and treatment of the various types of peritonitis are considered is of great interest.

Blood banking, the use of Mueller's solution and Elman and Weiner's method of giving amino acids for protein replacement are described. Clute's incision is given for exposing the diaphragm, the terminal esophagus, and the cardiac end of the stomach. A new tube gastrostomy is described which may be used as a substitute for the intranasal Jutte or Levine tube.

The treatment of appendicitis is considered in great detail, and the most recent statistics are given.

Operations for lesions of the intravertebral discs and hypertrophy of the ligamentum flavum are described and illustrated, as also is the treatment for bronchial asthma by division of the sympathetic and parasympathetic nerves in the root of the lung.

The section of plastic surgery is especially well illustrated. Operations for webbed fingers, the repair of harelip and cleft palate deformities, descriptions of pharyngoplasty, operation for ptosis of the eyelids, operations for rhinophyma, and the tubed pedicle flaps are described and illustrated.

In the section on orthopedics the new technic for nailing intracapsular fractures of the hip, a bone peg for ununited fractures of the carpal scaphoid, Schanz's osteotomy, McMurray's osteotomy, Stein's operation for hallux valgus, a new operation for hammer toe, operations to equalize the length of the lower extremities, Ober's fasciotomy for sciatica, an operation for unstable knee, Smith-Peterson's incision for exposure of the hip, Campbell's new shelf operation at the hip, and a new operation for acromioclavicular dislocation are described.

Gynecologic surgery is not touched upon in this work.

H. J. PRENDERGAST, M.D.

NEW AND NON-OFFICIAL REMEDIES, 1940. Containing descriptions of the articles which stand accepted by the Council on Pharmacy and Chemistry of the American Medical Association on Jan. 1, 1940. Cloth. Price, postpaid, $1.50. pp. 656-LXVIII. Chicago: American Medical Association, 1940.

Each year a revised list of the articles which stand accepted by the Council on Pharmacy and Chemistry of the American Medical Association as of January first is published in book form under the title of "New and Non-official Remedies." The book contains the descriptions of acceptable proprietary substances and their preparations, proprietary mixtures if they have originality or other important qualities, important non-proprietary non-official articles, simple pharmaceutical preparations, and other articles which require retention in the book.

A list of articles and brands accepted by the Council, but not described, is included in the book to cover simple preparations or mixtures of official articles (U.S.P. or N.F.) marketed under descriptive, non-proprietary names for which only established claims are made. Diagnostic reagents which are not used in or on the human body, and protein diagnostic preparations are not included in New and Non-official Remedies unless the determination of the status of these products by the Council has been requested by the distributor: If such products are found to be marketed in accordance with the Council's rules, they may be included in the list of undescribed, but acceptable articles.

New and Non-official Remedies is a practical and condensed text of pharmacology and therapeutics; it contains scientifically elaborated standards for all accepted non-official drugs; its Index to Distributors is a list of manufacturers, a large number of whose products have met the Council's high standards; its Bibliographical Index is a storehouse of references to reports which have been made mainly on unaccepted and unacceptable drugs; its prefatory material contains the

Council's "Rules," a time-tested and reliable set of basic principles for the furtherance of scientific and rational medicine.

A supplement to the annual volume of New and Non-official Remedies is published twice a year to bring up to date such current revisions and additions as have been necessary since its last publication. Every product included in the book is subject to the official rules of the Council. The comments to rules are changed occasionally by way of clarifying interpretation to insure fair consideration of all submitted preparations as new standards are recognized. Such constant and critical consideration of its contents provides the physician with a valuable reference list of acceptable new preparations on which to base his selection for use in treatment according to the established current practices of the profession.

The 1940 New and Non-official Remedies, of course, contains the revisions which appeared in the supplements for the 1939 edition, and continues the plan of grouping together articles having similar composition or action under a general discussion. These discussions have undergone considerable revision in the 1940 edition. Further revision of statements regarding the actions, uses, dosage, composition, purity, identity, strength or physical properties of many of the articles has also been necessary in some cases. Noteworthy revisions are those of the chapter on Liver and Stomach Preparations, radically rewritten and including a statement of requirements suggested by findings of the Anti-Anemia Preparations Advisory Board of the U. S. Pharmacopeia; the subsection Tuberculins, entirely rewritten to conform to newer knowledge in this field; and the chapter Allergenic Protein Preparations, the name of which has been changed to Allergenic Preparations. Minor but relatively important revisions are found in the articles: Bismuth Compounds, Serums and Vaccines, and Vitamins and Vitamin Preparations for Prophylactic and Therapeutic Use.

The indices of the new volume of New and Non-official Remedies are of the same order and plan as in previous editions. A general index lists accepted articles, including those not described. This is followed by an index to distributors in which appear all the Council accepted articles listed under their respective manufacturers. Finally, a bibliographical index is added for listing proprietary and unofficial articles not included in N.N.R. This includes references to the Council publications concerning each such article as has appeared in *The Journal of the A.M.A., Reports of the Council on Pharmacy and Chemistry, Propaganda for Reform, Vols. 1 and 2,* or *Reports of the A.M.A. Chemical Laboratory.*

HISTAMINASE IN THE TREATMENT OF URTECARIA OF PREGNANCY

(Continued from page 797)

cluding phenobarbital, bromides, nembutal were used in large doses to alleviate the marked distress of the patient who, by this time, was getting very desperate. The condition became so severe that therapeutic abortion was seriously considered as the patient's mental condition was steadily deteriorating and the skin was one mass of urticaria wheels and excoriations.

On March 11, 1940, histaminase was tried. Ten units were given with water four times a day before meals. Within twenty-four hours the skin became quite clear and the itching a great deal alleviated though not entirely gone. By keeping on this regime, a fair amount of comfort could be obtained. On March 27, 1940, the patient had a painless hemorrhage. A diagnosis of placenta previa was made and a cesarean section was performed by Dr. A. Hoaglund. Twin girls were delivered without complications and a central placenta previa was found. Immediately after delivery, the urticaria and itching disappeared entirely though the histaminase was discontinued just previous to operation.

Comment

A case is reported of urticaria of pregnancy which was apparently helped by histaminase after all other means tried had failed to give any relief.

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